The Effect of Written Emotional Expression on Depression Following mild Traumatic Brain Injury: A Pilot Study

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Dedications

This research is dedicated to:

- my beloved grandmothers, Pinkie and Annie-Mae, who were beautiful, intelligent women who inspired me to pursue superior life goals—despite challenges;

-my parents, Karen and Edward, who provided me every opportunity to explore and learn (especially my mother who enthusiastically cheered me on in all of my endeavors);

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Abstract

The Effect of Written Emotional Expression on Depression
Following a mild Traumatic Brain Injury
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The purpose of the following study was to examine the effects of an emotional expression exercise on depressed mood in a population of individuals who sustained mild Traumatic Brain Injury (mTBI). Depression is the most common psychological disorder that develops following mTBI and a significant number of people are affected by it. It was proposed that emotional expression could be a practical, cost-effective, and time-efficient tool that might decrease depressed mood in this population. Forty-two participants with history of mild Traumatic Brain Injury and who reported experiencing depressed mood were recruited for this study. Participants were divided into two groups, Emotional Expression (EE) and Neutral (N) groups, and were required to complete three writing exercises from their homes. The Emotional Expression group was instructed to write about their thoughts and feelings about their head injury. The Neutral group was instructed to write about the details of the day, excluding thoughts and feelings. Each participant also completed measures of immediate affect, mood, coping, and neurobehavioral functioning. Low power prevented this study from obtaining significant results in most of the outcome measures. Relationships between depressed mood and three coping indices (behavioral disengagement, planning, and acceptance) were revealed. the null hypothesis, Despite some findings, statistical analyses failed to capture or disconfirm hypotheses. Therefore, a post-hoc qualitative analysis was accomplished. Responders were compared to non-responders and differences in narratives emerged. Analysis revealed that focusing on perceived benefits may

result in a reduction in symptoms associated with depressed mood following mTBI. Implications for future research in this area are discussed.

Chapter 1: Introduction

Two million individuals sustain mild Traumatic Brain Injuries (mTBI) in the United States every year. Currently, it is estimated that there are over 5 million people living with history of mTBI in the U.S (Martin, Lu, Helmick, French, & Warden, 2008; Varney & Roberts, 1999). Approximately one third of this group report persisting symptoms associated with head injury over a protracted period (Kile, 2006; Ruff, Camenzuli, & Mueller, 1996). Given that the highest incidence rates of mTBI occur between the ages 15-24, the medical community is faced with a population of people who have sustained brain injuries, yet are expected to have significant portions of their life spans remaining (Barth, Alves, Ryan, et al., 1989; Lezak, Howieson, Loring, Hannay, & Fischer, 2004). More specific to the current research proposal, it is estimated that up to 70% of individuals develop depression following a head injury (Alderfer, Arciniegas, & Silver, 2005; Aloia, Long, & Allen, 1995; Busch & Alpern, 1998; Kreutzer, Seel, & Gourley, 2001). In fact, research suggests that individuals who sustain mTBIs are at higher risk for depression than those who sustain more severe brain injuries (Varney, Martzke, & Roberts, 2002). Due to the considerable number of individuals who sustain mTBIs and who continue to require medical and psychological services for many years following their injuries, the burden on the health system is immense. This phenomenon is magnified in the United States military, as service members are at increased risk of sustaining mTBIs during war or hostile conflicts. 22% of soldiers wounded in Afghanistan and Iraq sustained mTBIs. As of 2006, 1.4 million service members were reported to have suffered mTBI due to blast injuries (Hoge, McGurk, Thomas, Cox, & Castro, 2008; Martin, et al., 2008). As a result, it is important to develop and investigate treatment strategies that are cost-effective and therapeutically efficacious, but will not present undue burden to the health system.

Treating depression following mTBI is vital to the functioning of individuals who not only appear to have diminished coping ability, but who also subjectively report persisting symptoms of head injury. The impact of depression extends past the significant decrease in the

quality of life for many who experience depressed mood in months and even years following their head injuries. Depression is also likely to result in significant burden to the medical community. For example, contact between the individual who sustained mTBI and medical providers/specialists significantly increases. This results in substantial healthcare costs. It is reported that, as a result of the number of mTBIs sustained and the potential persisting sequelae that ensue, mTBI costs the United States approximately 17-38 billion dollars annually (Bazarian, Mcclung, Shah, Cheng, Flesher, & Kraus, 2005; Centers for Disease Control, 2003). In the context of the military, the healthcare costs are also significant. Additionally, mTBIs may result in loss of well-trained personnel.

Several additional themes are documented in the head injury literature that support the importance of treating depression following mTBI: (a) a significant number of people sustain mTBI in the United States alone; (b) mTBI sufferers are at greater risk for developing depression than severely head-injured individuals; and (c) if untreated, it is possible that depression complicates the recovery process from head injury. Despite the fact that depression among head-injured individuals may pose a significant drain on the public health system, empirical support for effective treatment strategies for depression that have been investigated following mTBI sufferers is sparse (Alderfer**,** Arciniegas, & Silver, 2005). Therefore, developing and investigating treatment for depression following mTBI is important, as a significant group of people may experience complications that require overwhelming attention from the medical and mental health communities.

The relationship between depression and persisting symptoms of mTBI is not well understood. Symptoms of depression and head injury overlap, making it difficult to distinguish depression from self-reported post-concussive symptoms. Prolonged symptom experience may also be reflective of a more severe head injury than initially diagnosed. Depression likely contributes to complications in recovery from mTBI. Whether or not depression significantly compromises neuropsychological functioning is controversial (Fann, Uomoto, & Katon, 2001;

Rohling, Green, Allen, & Iverson, 2002; Satz, Forney, & Zaucha, 1998). Nevertheless, it is well documented that depression among the head-injured may contribute to poor quality of life, including delayed return to work, difficulty in family and social interactions, overall perceived compromise in functionality, and increased difficulty in activities of daily functioning (Busch & Alpern, 1998; Miller & Mittenberg, 1998).

Despite the importance of treating depression following mTBI, research studies supporting effective treatment for depression among individuals with mTBI are few (Alderfer, Arciniegas, & Silver, 2005; Ragnarsson, 2006) . Some advances have been made in the area of psychopharmacology, as several studies have investigated the effects of antidepressants on depressive symptoms in head-injured populations. However, these studies yield mixed results (Alderfer, Arciniegas, & Silver, 2005; Fann, Katon, Uomoto, & Esselman, 1996; Fann, Uomoto, & Katon, 2000; Fann, Uomoto, & Katon, 2001; Kile, 2006; Mobayed, 1992; Mobayed & Dinan, 1990; Newburn, Edwards, Thomas, Collier, Fox, & Collins, 1999). Additionally, the cost of medication may be great over the long term, despite such variability in treatment effectiveness. Researchers encourage the use of behavioral and cognitive-behavioral psychotherapeutic methods to treat depression subsequent to mTBI (Alderfer, Arciniegas, & Silver, 2005; Busch & Alpern, 1998; Onswerth & Oei, 1998; Raskin & Stein, 2000). However, there is little empirical support for the effectiveness of these techniques to reduce depression following mTBI. Both psychopharmacologic and psychotherapeutic treatment models may prove to be promising for treating depression following a head injury, as these are the treatment options for the non-head-injured population. Although the mechanisms responsible for depression following mTBI are not completely understood, it is possible that these mechanisms are similar to those in the non-head-injured population. This is assumed because, despite the possible causes of depression, the spiraling effects of depressed mood eventually involve a compromise in biopsychosocial factors that are amenable to psycho- and pharmacotherapies. However, identifying and better

understanding the specific mechanisms that may underlie depression following mTBI may result in a treatment strategy that specifically targets this population.

The links that exist between depression and post-concussive syndrome (PCS), which describes a pattern of symptoms that may develop following mTBI, may help in developing an effective treatment model. Research aimed at preventing and treating PCS following head injury suggests that psychosocial methods are effective (Miller & Mittenberg, 1998). As both PCS and depression may develop as a result of inadequate coping methods in response to environmental stress that is experienced following a head injury, implementation of better coping strategies and expectation setting, an alternative model to conventional psychotherapy and pharmacotherapy, may be useful in treating depression effectively. For instance, educating patients shortly after they have sustained mild head injuries has been connected to lower PCS rates (Alves, Macciocchi, & Barth, 1993; Ponsford, 2002). Understanding the appropriate course of injury is expected to help patients cope with their head injuries. While psychoeducation can result in increased capacity to cope by reducing the likelihood of persisting report of head injury symptoms, other psychosocial methods that target coping, for instance, through emotional self-regulation, may also result in a reduction of depressive symptoms.

The emotional expression paradigm (Pennebaker, 1986) may be particularly useful for reducing depression among individuals who have sustained mTBI. Written emotional expression has been found to increase coping ability in a number of healthy and medical populations (for review, see Pennebaker, 1998; Smyth**,** 1997; Stanton, Danoff-Burg, Sworowski et al., 2002). In these studies, writing about a traumatic event over three to four occasions resulted in improved health and health behaviors. Specifically, L'Abate et al. (1996) observed a decrease in scores on depression measures following an emotional expression paradigm. Perhaps this paradigm will be useful in decreasing depression following an mTBI. The following proposal will briefly review mTBI, including diagnostic methods and depression following mTBI. More specifically, incidence, course, theories about etiology of depressive symptoms, and methods of treatment of

depression will also be reviewed. Finally, the use of emotional expression to decrease depression symptoms following an mTBI will be proposed.

Chapter 2: Review of Incidence and Diagnosis of mild Traumatic Brain Injury (mTBI)

The role (i.e., etiology, prevalence, and course) of depression following mTBI is not well understood. The contribution of organic versus psychosocial factors that contribute to the cascade of symptoms that follows mTBI is equivocal. As a result, delineating psychological consequences from sequelae related to an organic brain injury may complicate treatment methods. To further clarify the complexity in diagnosing and treating depression following a mild head injury, a basic review of the prevalence of mTBI and how it is diagnosed is presented in the following section.

Incidence

Mild traumatic brain injuries comprise the greatest proportion of all head injuries, with estimates ranging from 325,000 to more than 2 million incidences annually (Martin, et al., 2008; CDC, 2003; Varney & Roberts, 1999). Large variations in the rate at which mTBI occurs and the uncertainty about expected recovery course are largely due to factors that arise when assessing the severity of a head injury in clinical research studies (Ragnarsson, 2006; Varney & Roberts, 1999). For example, review of this body of literature revealed common issues including the lack of scientific and biological evidence, inconsistent criteria used for diagnoses, and unreliable subjective reports of symptoms and neurobehavioral data at the time of injury (Malec, 1999; Ruff, 2005; Uzell, 1999). The inconsistencies noted in these clinical studies possibly contribute to the confusion that ensues over whether the severity of head injury captured in these investigations is mild or severe, which makes it difficult to parcel out how depression may affect the recovery process, using this body of research. Perhaps persisting symptoms of head injury are less related to any complications caused by depression and are more related to organic disruption. This is important in that an individual with a head injury may not be referred for appropriate medical or psychological treatment.

Defining mild Traumatic Brain Injury (mTBI)

There is no universally accepted definition of Traumatic Brain Injury (TBI). Definitions typically reflect the general idea that TBI is a "broad range of neurological, cognitive, and emotional factors that result from the application of a mechanical force to the head" (p. 1, Kibby and Long, 1996). Mechanisms of force can include a hit to the head, the head striking against something (e.g., following a fall), acceleration/deceleration forces, and traumatic incidents during which the head is not struck (e.g., electrocution, shock waves, etc.). The severity of TBI is assessed as mild to severe based on the actual physiological impact of the mechanical force on the brain. This force may result in lesions, diffuse axonal injury, metabolic disruption, and/or vascular injury, which may be captured in neuroimaging or through neurobehavioral assessments. More specifically, mTBI is often diagnosed when there is evidence of brain disruption that results in either mild or no physical findings (Kibby and Long, 1996). Other terms used to define TBI, for instance, minor head injury, concussion, closed head injury, and mild head trauma, have been used by researchers and clinicians. Some may suggest that the differences among these terms are great, while others assert that there are small gradations of differences along the concussion to mTBI continuum. For example, a closed-head injury is reflective of a trauma to the head without any objects penetrating through the skull. The severity level of closed-head injuries may range from mild (as in mTBI) to severe, depending on the level of damage to the brain. To take concussion as another example, it has been described as a temporary loss of brain functioning that quickly resolves. In the past, a loss of consciousness was one of the defining features of a concussion. Currently, it is accepted by many that any transient change in mental status may be reflective a concussion (Cajigal, 2007). Often, the term concussion is used interchangeably with mTBI. When someone is diagnosed with a concussion, residual symptoms are expected to be minimal; however, self-reported post-concussive symptoms have been reported to persist for some patients. In one last example, despite its name, minor head injury may encompass TBIs that are assessed to be mild to moderate, not just mild (Kibby and Long, 1996). Ruff (2005) indicated

that even the term head injury has been further defined, as head injury only denotes a trauma to the head, while brain damage would suggest that an actual injury to the brain occurred. These two, brain and head injury, may or may not co-occur. Despite this refined definition, clinicians and researchers still may not differentiate them from each other, which results in these terms being used interchangeably (Ruff, 2005). Many of these terms have been clarified at some point and may be intended to describe and reflect a specific phenomenon.

Despite ongoing clarification of these terms and "syndromes", they are not consistently used in the field and are often used interchangeably in research (Ruff, 2005). Ragnarsson (2006) reviewed goals established in 1998 and he concluded that the obtaining more uniformity in describing injury profiles and severity in empirical research has gone unaddressed. Further, Kibby and Long (1996), suggested that the vague subjective limits used to determine severity level and the failure to differentiate between cognitive versus post-concussive symptoms add to the confusion that exists in the mTBI literature. As noted by Kibby and Long (1996), it is also evident in the literature that the term mild in mTBI is not reflective of the severity of emotional or neurocognitive symptoms that may follow mTBI, as the severity of these symptoms may range from non-existent to so severe the patient's functioning is disrupted (Cajigal, 2007).

For the purposes of this study, where possible, clinical research investigating patients with mTBI was reviewed. In areas where empirical research was sparse, studies pertaining to TBI, in general, were used as guide to develop the rationale for the current study. The participants of the current study were invited to participate because they were reported to have history of mTBI, using the guidelines from the mild Traumatic Brain Injury Subcommittee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation (1993). In a military population, there is an increased risk for members to sustain mTBIs in combat. More specific to this study, blasts injuries comprised the greatest mechanical force, which is described as a high pressurized wave that is followed by a blast of wind that moves away from the explosion (shock wave) at supersonic speed. Similar to what is observed in non-blast

TBI, the mechanisms for sustaining TBIs include penetrating injuries to the head from debris and shrapnel, impact to the head from debris, the head's impact on another object (due to being thrown during the wave), and thermal and/or neurotoxic injuries. The pathophysiology of these injuries is not well understood (Martin, et al., 2008). However, current literature suggests that there is likely diffuse axonal injury, vascular, and metabolic damage. The US Army is currently testing helmets with sensors to better understand the impact of blasts, which they believe may lead to a diagnostic tool for TBI (Martin, et al., 2008). The improved and quick response by military medics may result in additional information from neuroimaging, which may also provide more data about the pathophysiology. The following will outline more specifically how mTBI is assessed and additional concerns that have been identified in the mTBI literature, in general.

Biological Evidence for mTBI: Neuroimaging

Computed Tomography (CT) is the most widely used neuroimaging technique in emergency room settings, partly because it is extremely sensitive to the possibility of a fatal intracranial bleeding, but more often because it is one of the more economical imaging techniques compared to other forms of imaging (Lezak, et al., 2004). CT scans reveal positive signs of mild brain damage in only 15-24% of mild head injury cases. In contrast, Magnetic Resonance Imaging (MRI) is more sensitive to detecting non-hemorrhagic lesions, diffuse axonal injury, and smaller contusions, which would presumably result in a higher number of definitive findings in neuroimaging studies immediately following a minor insult to the brain (Varney & Roberts, 1999). Nevertheless, very rarely are both of these imaging techniques used in tandem following a minor insult, and CT continues to be the preferred method of imaging following mild head injury. Thus, the greater use of CT as a means of determining the likelihood of brain injury likely results in a higher number of head injuries diagnosed as mild in severity, as opposed to moderate to severe.

The possible underestimation of head injury severity, based on neuroimaging data, could result in a poor understanding of the contribution that depression has to the recovery process.

Perhaps the individual who sustained the head injury is not following the expected recovery course, not as a consequence of depression, but because the injury was actually more severe than initially diagnosed. Moreover, the organic compromise may contribute to the depression more than poor coping ability or feelings of loss due to decreased cognitive ability. These factors may result in treatment that is not appropriate for the individual, resulting in prolonged and/or ineffective use of health or clinical services. Even if poor coping ability is not related to subjective post-concussive complaints, the currently proposed intervention may prove useful in improving the individual's ability to identify and adjust to the changes in his or her functioning following head injury.

Head injuries that are graded as mild are often diagnosed as such because physical evidence from imaging studies immediately following insult to the head fails to identify structural damage. Although Hayes and colleagues (1999) suggest that subtle physiological occurrences in mild brain damage possibly results from neurochemical abnormalities, most studies employed immediately following injury only measure structural changes (e.g., Computed Tomography). Consequently, a more severe head injury may not be suspected due to lack of objective findings. Further, even positive findings for structural damage may not result in significant long-lasting, functional deficits. Nevertheless, any and all structural and neurophysiological changes resulting from a mild head injury are purported by some to be minimum or even reversible (Hayes, 1999).

Neurobehavioral Criteria for mTBI

Physical evidence to support the diagnosis of mTBI is often lacking. As a result, diagnosticians rely on neurologically mediated behaviors to determine the presence and severity of a traumatic brain injury, such as loss of consciousness (LOC) or post traumatic amnesia (PTA) (Hayes, 1999; Ruff, 2005). Previous criticism of the use of neurobehavioral criteria to document head injury was that clinicians used inconsistent guidelines from diagnosis to diagnosis, resulting in misdiagnoses. Thus, a patient who was expected to recover in approximately three months might continue to complain of persisting symptoms legitimately due to a more severe injury that

was not identified at the time the effects of the injury were evaluated (Bigler, 2001). In attempts to obtain uniformity in diagnosing mild head injury, the mild Traumatic Brain Injury Subcommittee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation (1993) devised criteria to assess mild traumatic brain injury. As outlined in Horn (1992), it was proposed that the presumed head-injured individual manifest one of several symptoms:

> The patient must have sustained, following the injury: 1) any period of a loss of consciousness (LOC); 2) post-traumatic amnesia (PTA)-retrograde or anterograde; 3) an alteration in mental state (i.e. confusion, disorientation, or daze); 4) a focal neurological deficit in absence of LOC greater than 30 minutes; 5) Glasgow Coma Scale Score (a scale that measures one's level of consciousness) that is less than 15 after 30 minutes; 6) and/or PTA no greater than 24 hours. (p. 5).

These criteria have resulted in greater cross-comparison in research. Nevertheless, the low frequency of positive imaging findings indicating that brain damage occurred following mTBI fuels the debate over the legitimacy of the mechanisms responsible for neurobehavioral changes. Moreover, positive neurobehavioral findings at the time injury is diagnosed are likely to be considered reversible, and as a result, insignificant. Thus, persisting symptoms are likely attributed to the symptoms associated with depression or other psychological disturbances, and are not deemed the direct result of an organic insult. As stated before, it is asserted that organic compromise may, in fact, underlie the symptom constellation that emerges following the inaccurately assessed head injury (Bigler, 2001). As the etiology of depressive symptoms is unclear following mTBI, if in fact it is found that depressive symptoms are organically mediated, the proposed intervention may not reduce depressive symptoms. In this case, physical symptoms of depression may be permanent and may not be reflective of temporary compromise in emotional functioning. Instead, a change in coping ability may emerge following the exercise. Further, a change in cognitive style may be evident. Should depressed mood arise from poor coping ability, the expectation is that the exercise undertaken in the current study will improve coping ability, thereby improving depressed mood.

Self-reported Symptoms following mTBI

A number of symptoms are expected following mTBI. These symptoms are often elicited through self-report. Subjectively reported sequelae include attention and concentration difficulties, memory dysfunction, dizziness, sleep disturbance, headache, sensitivity to light and sound, blurred vision, tinnitus, and neck ache (Kile, 2006; Yang, Tu, Hua, & Huang, 2007; Varney & Roberts, 1999). These symptoms range in severity irrespective of head injury severity. The etiology of these symptoms, (i.e., fatigue, headache, concentration difficulties, etc.) are vulnerable to ambiguity when comparing a head-injured group to any other medical population. Nevertheless, in a controlled study, Paniak and colleagues (2002) revealed aspects of post-concussive symptomatology (i.e., increase in cognitive complaints over time and depressed mood) that were unique to mTBI patients when these patients were compared to patients without mTBI.

Inasmuch as the resulting cognitive and somatic complaints are residual of the mTBI sustained, spontaneous recovery of cognitive functioning is expected within three months to one year post-injury for many mTBI sufferers (Szymanski & Linn, 1992). Mild trauma to the brain and symptoms associated with the trauma are reported by scientists and clinicians to be reversible. As such, most functions have been reported to return to normal within weeks following injury (Yang, Tu, Hua, Huang, 2007). This excludes the recovery of attention and concentration and problem solving abilities, which may recover in up to one year's time, if affected (Lezak, et al.,2004). Notwithstanding these expectations, approximately 33% of individuals sustaining mTBI continue to complain of injuries beyond the expected recovery period (Dikmen, Ross, Machammer & Temkin, 1995; Ruff, 2005; Varney & Roberts, 1999; Yang, Tu, Hua, & Huang, 2007). Of this group, a portion of these patients claim persisting cognitive problems many years following their injury, which is counter to expectation following mTBI. Patients with residual impairment are diagnosed with post-concussive syndrome (PCS), a

syndrome characterized by post-concussive symptoms failing to resolve within the expected period of time (three months to one year).

Following the expected recovery period from mTBI, some clinicians may question the legitimacy of the persisting effects of the injury sustained. These lasting residual effects of mTBI are often called into question because of the lack of physiological evidence, resolved neurobehavioral deficits, and the heavy reliance on patients' subjective reports. Debate ensues with respect to the legitimacy of the persisting cognitive complaints by mTBI patients. Moreover, this population is often associated with litigation, which raises questions about exaggeration of symptoms and protraction of symptom course for secondary gain (financial compensation or relief from work) (Heubrock & Petermann, 1998; Iverson & Binder, 2000; Lynch, 2004; Richman, Green, Gervais, Flaro, Merten, Brockhaus, & Ranks, 2006). However, the expected course following mTBI continues to be investigated and is not completely clear. More specific to this proposal, the course and etiology, and effects of psychological sequelae of mTBI, like depression, are also unclear. What is clear is that depression is a significant syndrome that emerges in those within the mTBI population who report persisting symptoms. Therefore, the proposed study was twofold in purpose: 1) to develop a strategy to reduce depression using a psychosocial intervention developed to target affective coping (drawing from various methods developed to address depression, neuropsychological sequelae of mTBI, and other medical populations studied in clinical health psychology); and 2) to determine the effectiveness of this strategy for people who sustain mTBI and who report depressed mood.

Etiology of Post-Concussive Syndrome (PCS)

The cause of persisting post-concussive complaints following the expected recovery period following mTBI is not clear. Theories range from factors that are organic to those that are psychogenic. Others propose theories that take into consideration each of these factors among a number of individual differences (age, premorbid psychiatric history, etc.) (Bigler, 2001; Cicerone & Kalmar, 1997; Ruff, Carmenzuli, & Mueller, 1996). Persisting complaints could

reflect residual effects of the injury that have yet to resolve. Regarding organic causes, Bigler (2001) and colleagues suggest that more severe damage might have occurred in the brain than initially diagnosed. Moreover, the type of imaging studies employed at the time of injury may have been insufficient in detecting this compromise in neural tissue or neural activation. Also, residual hemorrhages following discharge from hospital care and/or tiny undetectable lesions may be responsible for the persisting cognitive problems (Varney & Roberts, 1999). However, these views are less supported than the multifactorial and psychogenic theories, likely due to the difficulty in assessing such minuscule disruptions in metabolic functioning and possible functional neural changes.

Alves and colleagues (1993) among other researchers (Kay, 1992) describe mTBI as a multifactorial disorder. Actual brain damage is not assumed to be the only factor responsible for persisting deficits. Rather, many factors, such as the effects of age, level of education, type of cerebral trauma, premorbid brain integrity, and psychological consequences of the incident are presumed to contribute to the post-concussive complaints. This theory is inclusive of the many factors, including the amalgamated affect of organic changes, psychosocial stress, and psychological consequences, that likely contribute to the effects observed following mTBI.

Other researchers attribute the persisting deficits and post-concussive symptoms to psychogenic etiology, mostly acute distress and depression following the injury (Cicerone & Kalmar, 1997; Ruff, Carmenzuli, & Mueller, 1996; Varney & Roberts, 1999). This is a likely attribution as a high incidence of depressive symptoms is commonly found in the mTBI population. Therefore, understanding the role of depression with respect to individuals who have sustained mTBIs is valuable, as depression is likely to impact the overall quality of life for a significant number of people. More importantly, investigating an effective treatment model would help to parcel out the mechanisms responsible for persisting symptom report. Regardless of the cause of depression (organic or reactive), it is important to identify depression following mTBI so the appropriate treatment recommendations are made. The following section will review the

prevalence, course, and etiology of depression, based on current theories, following head injury, specifically mTBI. Current treatment strategies for depression following mTBI will also be reviewed.

Chapter 3: Prevalence of Depression Following Head Injury

According to the literature, depression is the most commonly found emotional disturbance following any head injury, especially following mTBI. The value of understanding how to identify and treat depression is immense, as depression may contribute to decreased quality of life in the long-term for individuals who sustain mild head injury (Busch & Alpern, 1998; Kile, 2006; Kit, Mateer, & Graves, 2007; Ruff, Camenzuli, & Mueller, 1996). Persisting depression may also contribute to an increased risk for suicide in any population. Moreover, investigation of the effects of depression following mTBI is valuable to the clinical community, as traumatic head injury remains the most common cause of brain damage and is likely to be encountered by health care professionals.

Research findings over the last two decades support that head injury results in an increased risk for developing depression (Kile, 2006; Kit, Mateer, & Graves, 2007; Schoenhuber & Gentili, 1988). Several researchers empirically investigated the prevalence of depression, using well-validated measurements, and it was found that 7% to 70% of individuals who sustained head injuries reported some level of depression. (Alderfer, Arcinieags, & Silver, 2005; Alexander, 1992; Ettlin, Kischka, & Reichman, 1992; Fann, Katon, & Uomoto, 2000; Kreutzer, Seel, & Gourley, 2001; Parker & Rosenblum, 1996; Schoenhuber & Gentili, 1988). This range is quite large. The variability is likely due, in part, to the manner in which depression is assessed in these studies. For instance, several studies reported using self-report inventories or one-item questionnaires, while others reported using criteria outlined by the DSM-IV and structured clinical interviews (Busch & Alpern, 1998; Rohling, Green, Allen, & Iverson, 2002). As depression and head injury share many common symptoms, some tools are likely to underestimate or overestimate depression, based on the individual's report (Sawchyn, Brulot, & Strauss, 2000; Woessner & Caplan, 1995). Moreover, this disparity may also be due to the frequency and severity of self-reported symptoms, which may be a function of the severity of the

head injury. More severe injuries may result in anosagnosia, which would likely result in the underreporting of symptom experience. Alternatively, mild injuries sustained by high functioning individuals could result in increased awareness of change, which might result in overestimation of symptom experience.

Other contributing factors to the disparate rates in the prevalence of depression following head injury are the various methodological differences among research studies (e.g., time since injury, onset of depression, and type of injury, among other factors). For instance, in one study, Schoenhuber and Gentili (1988) found that of mTBI patients, 39% were depressed at 12 months following their head injuries. Although the percentage of depressed, head-injured patients was significantly higher than the incidence of depression in matched controls who suffered orthopedic injuries, the researchers in this study used a simple mail-in survey and depression checklist, which are vulnerable to the previously mentioned factors. Since the time Schoenhuber and Gentili's study was published, several studies have employed rigorous research designs and validated measures to reduce the errors that often limited the findings of studies that investigated depression following mTBI (see for instance Kreutzer, Seel, & Gourley, 2001).

In one such study, Kreutzer, Seel, and Gourley (2001) found that using the checklist derived from the DSM-IV in conjunction with the Neurobehavioral Functional Inventory (NFI), resulted in more reliable prevalence rates of depression following head injuries of varying severity. In this study, they attempted to control for common methodological errors, including the use of a highly reliable and valid measure to assess depression, a larger sample size, heterogeneity of head injury severity, and more diverse time since injury. By doing this, their study provided a more comprehensive profile of the rate of depression following traumatic brain injury. Kreutzer, et al. (2001) reported that 42% of their head injury sample met the criteria for a major depressive episode, with more than 20% of the participants reporting symptoms in seven of

the nine domains. Their findings were similar to that of Jorge and colleagues (1993) who also used the DSM-IV to assess depression following a head injury.

Based on the findings of these researchers combined, including the base rate of depression following head injury, it is reasonable to assume that depression is a worthy topic to investigate in the mTBI population. Furthermore, it is necessary to validate an effective treatment strategy in this population. However, the tool used to assess depression among the head-injured may be vulnerable to factors unique to this population (Sawchyn, Brulot, & Strauss, 2000; Woessner & Caplan, 1995). For instance, Woessner and Caplan found that scores on the SCL-90-R were elevated in a head-injured population when compared to a non-clinical, non-head-injured population. They hypothesized that this elevation was likely due to the overlapping symptom constellation shared by both depression and PCS. Sawchyn, Brulot, and Strauss (2000) arrived at a similar conclusion as a result of their study findings that suggested a relationship between scores on a post-concussion syndrome checklist and a closed-ended depression measure. As such, a measure that has been empirically validated in a head-injured population, or one that utilizes a format that mirrors a clinical interview, would likely be more effective at assessing depression than a brief symptom inventory.

One such measure, the Structured Assessment of Depression in Brain Damaged Individuals (SABD), was developed by Gordon and Hibbard (1997) to assess depression following stroke. The SABD is a measure with items taken from the Beck Depression Inventory (Beck, 1976) and the Hamilton Rating Scale for Depression (Hamilton, 1960) with somatic and emotional variables loading on separate indices. This measure was found to be highly reliable and valid in a post-stroke population. As this neurologically impaired population presents with similar symptoms as the mildly head-injured population, Raskin and colleagues suggested that this measure be utilized in a traumatic head-injured group as well (Raskin & Stein, 2000). Another measure, as mentioned before, is the Neurobehavioral Functional Inventory (NFI) (Kreutzer,

Seel, & Gourley, 2001), which also appeared to yield more reliable prevalence rates of depression when compared to commonly used closed-ended depression inventories. The reason this measure yields higher reliability than that of standard depression measures is that the items on this measure were specifically investigated using a brain-injured population as the normative sample. Appropriately identifying depression in a head-injured population is of great importance to the current study. So, understanding the limitations of commonly used self-report inventories and the importance of measures that are reliably used to assess depression following an mTBI is necessary. For the current study, the NFI was used to measure symptoms associated with depression. As stated previously, this measure has been found to yield reliable prevalence rates of depression in a head-injured population. Unlike commonly used depression checklists and inventories, the NFI includes a number of items that assess mood and depressed cognitive and behavioral states (hopelessness and worthlessness). Although assessed by this measure, somatic symptoms are not as heavily loaded on this measure's depression index.

Course of Depression Following Head Injury

With respect to the course, irrespective of the severity of the head injury, the onset of depression may occur in short time following the head injury and may persist for an extended period post-injury. Federoff, Starkstein, and Forrester (1992) documented the earliest time at which depression begins. They found that head-injured patients admitted to a trauma unit following a traumatic brain injury experienced major depression as early as one month following head injury. They also found that 27% of the patients who were not depressed at one month post-injury reported symptoms indicative of major depression at one year.

Varney, Martzke, and Roberts (2002) found that the onset of depression occurred six months post-injury for 46% of their sample. Gerber and Schraa (1995) found a similar time of onset of depression to Varney and his colleagues. In their study, Gerber and Schraa compared mTBI patients to patients with only orthopedic injuries on the presence and severity of post-

concussive symptoms. They found that the report of depressive symptoms was a unique marker for patients who sustained head injury, as compared to the patients with orthopedic injuries. To further elaborate the findings of their study, Gerber and Schraa (1995) found that the report of depressive symptoms increased only in the group of mTBI patients by a six-month follow-up period.

Based on the literature, depression may persist past the period of expected recovery (three months to one year) in head-injured individuals. For example, Varney, et al. (2002) documented in their study that 77% of closed-head injury patients reported at least six depressive symptoms at two to eight years post-injury. This finding may suggest that depression is not organically mediated, but a reactive depression that develops following a diminution of coping abilities. However, as these studies were not longitudinal, they should not be used to create definitive markers for the earliest time at which depression develops or the latest time to which depressive symptoms persist. Rather, they document that depression may be a factor throughout the entire recovery course from a mild head injury and beyond.

Etiology of Depression following an mTBI

Aloia's Two-model Theory: Organic Brain Injury versus Maladaptive Coping

Aloia and colleagues (1995) presented two models that may explain why depression develops following an mTBI: (1) organic compromise to the brain and (2) "remorse" resulting from cognitive decline (p. 576). The first part of this model, organic compromise to the brain, suggests that depression may be the direct result of the head injury. That is, physiological symptoms of depression result from actual brain damage. For instance, an organic depressive disorder may develop following an insult to the brain. The temporal and frontal poles of the brain are sensitive to injury in incidents of acceleration/deceleration (e.g., motor vehicle accidents), due to their proximity to the bony skull. Other researchers have implicated these areas in emotional functioning (Federoff, Starkstein, & Forrester, 1992; Jorge, Robinson, Arndt, Starkstein, 1993;

Ownswerth & Oei, 1998). However, research does not support a one-to-one relationship between the prevalence and severity of depression and neurobehavioral changes at the time of injury (i.e. length of LOC or PTA). Therefore, organic factors may not have the largest role in the majority of depressed individuals, especially following minor injuries to the brain. Moreover, objective findings of brain injury following head injury, as observed on neuroimaging, are rare. As a result, documenting a connection between the anterior regions of the brain and depression following an mTBI would be a difficult task.

Saran's (1985) research findings may lend little in support for Aloia's first factor, as it applies to an mTBI population. Saran (1985) utilized another approach to investigate the organic basis of depression following brain injury. Using the dexamethasone suppression test (DST), Saran measured the cortisone secretions in response to exogenous release of dexamethasone in psychiatric patients with endogenous depression and mTBI patients with depression. The literature suggests that psychiatric patients with endogenous depression often demonstrate dysfunction of the hypothalamic-pituitary axis (HPA). This dysfunction results in a disruption of the catecholamines and cholinergic metabolism. Consequently, the dexamethasone suppression test was found to be abnormal in a high percentage of patients with HPA dysfunction. In Saran's (1985) study, consistent with previous findings, a high percentage of depressed individuals in the control group demonstrated abnormal findings on the DST (91%). This percentage was compared to the 10% of abnormal findings on the DST observed in the minor head-injured patients. However, the rate at which abnormal findings occurred is similar to that found in a normal population, which suggests that there is no evidence of HPA dysfunction. This implies that while the clinical picture of depression following mTBI is similar to that of an exogenous depressed population, the etiology of the symptoms or the neural basis of the depression may not be similar. Nevertheless, organic factors cannot be completely ruled out. As indicated before, the severity of the head injury sustained may not be as clear in number of cases, due to diagnostic issues.

The second part of Aloia's two-factor model is that once a head injury is sustained, depression emerges as an emotional consequence due to disappointment about cognitive loss or increased environmental demand following the injury. This factor is supported by research conducted by Macniven and Finlayson (1993). In their study they reported that higher levels of distress, measured by the MMPI, were associated with greater awareness of intellectual impairments. Based on these results one might hypothesize that depression and other psychological disturbances may be reactive and are dependent on patients' perceptions of their changes in cognitive functioning or impacts these injuries had on their lives. Results of this study also concluded that patients who were less disturbed by their temporary impairment and decrease in functioning regained cognitive functioning in the appropriate time frame (Aloia, Long, & Allen, 1995). Although any neurobehavioral changes or decreases in cognitive functioning are thought to be temporary or reversible, the perception of loss by the patient possibly contributes to the development of depression. Stein's (1996) finding further supports this hypothesis. He found that patients who were educated on the various expected symptoms, including time expectations and recovery course, had shorter recovery times when compared to head-injured patients who were not informed. It is possible that poor understanding of the course of recovery impedes the patient's ability to cope and manage the stressors associated with the head injury. Furthermore, Stein's (1996) research suggests that poor understanding likely impacts the patient's perception of cognitive loss (and possible permanence of the loss), and, as suggested by Aloia et al. (1995), remorse may be the underlying factor contributing to depression. Perhaps temporary symptoms (and their impact) associated with mTBI trigger negative cognitive patterns. Consistent with a cognitive behavioral rationale for depression, these negative patterns reflect negative views of self, world, and future (Beck, 1999).

To further elaborate on the relationship between coping ability and depression, several other researchers have concluded that the individual's ability to cope with the insult itself is a factor in maintaining depression and post-concussive symptoms (Machulda, Bergquist, Ito, &

Chew, 1998; Mittenberg & Burton, 1994). In one study, Machulda and colleagues (1998) investigated the relationship between the level of perceived stress and symptom frequency in a population of students who had histories of mild head injury. The relationship between perceived stress and two factors, symptom frequency and severity, was found. Moreover, Machulda and colleagues (1998) also found the highest correlation between depression and perceived stress, compared to the other factors that were investigated.

In general, depression as a reactive emotional disturbance, as opposed to a consequence of physiological disruption, is well supported. However, it is also likely that stress may exacerbate a process that initially began as organic compromise to brain functioning. Bush and Alpern (1998) suggest that a "stream of anatomical, physiological, and chemical changes" (p. 105). following a head injury results in increased susceptibility to external stressors that results in decreased ability to cope. They went on to describe depression as a behavioral pathology that is "triggered" by stress, but is additive when considered in context of a mild head injury.

Curran, Ponsford, and Crowe (2000) also investigated the link between coping and depression following traumatic brain injury. To elucidate the coping strategies used by mTBI patients one to five years post-injury, Curran and her colleagues (2000) compared a traumatic brain injury sample to a sample of patients with orthopedic injuries on indices of depression, anxiety, and the type of coping strategies utilized by the patients in each group. These researchers not only wanted to clarify the relationship between depression and coping, but also the differences in the way that a neurologically impaired group appeared to cope with injury when compared to an orthopedically impaired medical sample. Curran et al. (2000) found that several behaviors that are indicative of poor coping correlate with poor psychosocial adjustment in a depressed head-injured sample. For instance, they documented that wishful thinking, self-blame, and avoidance were among the myriad of behaviors that appeared to differentiate depressed mTBI patients from orthopedic patients (Curran, Ponsford, & Crowe, 2000).

Ruff, Camenzuli, and Mueller (1996) found that a premorbid diagnosis of a depressive disorder appeared to be a contributing factor to the development of a depressive disorder following an mTBI. Perhaps poor coping skills prior to a head injury result in increased vulnerability to coping or adjusting to acute changes that may occur following an mTBI. Nevertheless, poor coping skills may not be unique to a head-injured population. In contrast to the controlled study conducted by Paniak and colleagues (2002), during which they found that cognitive complaints and report of depressed mood appeared to differentiate mTBI patients from medical controls, Curran and colleagues' (2000) found that the orthopedic controls in their study reported similar levels of distress and similar diminished coping skills as the mTBI patients in their study. This similarity in distress and coping between a head-injured sample and a medical control group is supported by findings of other researchers (Malia, Powell, & Torode, 1995; Moore & Stambrook, 1994). However, the findings of the study conducted by Curran and colleagues (2000) may not be fully reliable. For instance, the hospital stay for orthopedic controls was significantly higher than the hospital stay for the head-injured group. The two groups' sample sizes were significantly unequal. Also pain was not well controlled in their study's design. Consequently, the orthopedic group may have found it difficult to adjust to chronic pain, which may have influenced their coping and emotional distress. Finally, it may not be appropriate to generalize the findings of Curran and colleagues' (2000) study to the mTBI population, as this study included patients with severe head injury. However, the findings of this study provide a foundation to understand the possible contributory factors to the development and course of depression following a head injury.

Nonetheless, poor coping strategies are found to be correlated with high levels of depressed mood (Moore & Anderson, 2002). Considering that depressed individuals with a history of mTBI appear to utilize poor coping strategies, a treatment model that targets coping ability in a head-injured sample is likely to result in a decrease in depressed mood in this group.

Considering Aloia's (1995) two factor model and other researchers' findings that support either an organic or psychosocial basis to depression, an alternative approach to identifying the etiology of depression following head injury may lie in understanding the *course* of depression. Onswerth and Oei (1998) contend that a prolonged period between when an individual sustains a head injury and when depression develops may be indicative of a reactive depression, whereas depression that develops immediately following the head injury likely indicates an organic affective syndrome. In accordance with this theory, Varney, Martzke, and Roberts (2002) found that of the mTBI patients included in their study, 46% reported the onset of depression at six months or later.

Depression may initially arise from organic injury; however, in a mildly head-injured population, a preponderance of evidence suggests that psychosocial factors likely underlie depression following a head injury. Specifically, an individual's diminished ability to cope with head injury sequelae, perceived cognitive loss, and stress may be responsible for the emotional disturbance mTBI. Therefore, a treatment model that is designed to reduce stress and assist the mTBI patients in coping with their injuries might be effective at reducing depression following head injury.

Treatment Indications for Depression following mTBI

The importance of treating depression lies in the individual's safety, cognitive functioning, emotional well-being, and overall quality of life. The risk of suicide increases following head injury and is elevated even more with depression. Failing to treat depression following head injury could result in increased hopelessness and despair, which may lead to self-injurious or suicidal behavior (Beck, 1976; Beck, 1996). To illustrate, Varney, Martzke, and Roberts (2002) conducted a study in which more than half of the depressed head-injured patients in their study had frequent crying spells and reported suicidal ideation.

It is questionable whether neuropsychological functioning (e.g., memory, psychomotor functioning, and attention) is compromised due to depression. Several researchers found that

individuals with depression (with or without brain injury) performed more poorly on neuropsychological tests when compared to normal patients (Kopelman, 1986, Rohling, Green, Allen, & Iverson, 2002; Satz, Forney, Zaucha, Asarnow, Light, McCleary, et al., 1998). Kopelman (1986) found that head-injured patients who met criteria for major depression performed more poorly than controls on tests of recent memory, Nevertheless, the depressed group's memory functioning was higher than that of the brain-injured group.

More specific to mTBI patients, Fann, Katon, & Uomoto (2001) investigated the effect of pharmacologic treatment for depression on cognitive functioning. They conducted a single-blind, placebo run-in trial of sertraline with individuals who experienced major depression following a mild head injury. Patients' performances were assessed by comparing their scores on neuropsychological tests from the placebo trial to the scores obtained at eight weeks, following therapeutic doses of sertraline. Fann et al. (2001) found that in accordance with decreases in depression scores, scores on measures of psychological distress, and post-concussive symptoms decreased at eight weeks as well. They also found that psychomotor speed, flexibility, and retention of new memories improved following antidepressant treatment. The findings of this study may be limited. Fann and colleagues (2001) did not utilize a control group, which would have been useful to control for practice effects. The investigators re-administered all of the same memory tests without alternate forms at eight weeks. At that time, the "trend" toward improvement on psychomotor tests and tests of attention and mental flexibility that was identified may have resulted from the previous exposure only eight weeks prior to the study. Additionally, it is known that depression may affect neurovegetative functioning, including psychomotor functioning. So, this finding does not provide new insight or make a strong case for the effectiveness of sertraline in decreasing depression, thereby improving neuropsychological functioning. This study does not strongly support the notion that neuropsychological functioning is impacted by depressed mood. Additional research would be useful to clarify these findings.

Rohling, Green and colleagues (2002) asserted that depression has no effect on neuropsychological functioning. They concluded that many of the studies that investigated the differences between neuropsychological functioning of depressed head-injured patients and non-depressed head-injured patients lacked appropriate scientific methodology. As such, the results were often overstated or flawed. In Rohling and colleagues' (2002) study, they attempted to control for common methodological errors. In their study, comprehensive neuropsychological evaluations were administered to outpatients seeking disability compensation. Neuropsychological test results of patients whose performance on tests were valid and reliable (based on two objective tests of validity) were included in the study. Patients who obtained low scores on the measures of depression were compared to patients who had obtained high scores on these same measures. Using statistical analyses that compared between group differences and correlations between scores on the depression measures and neuropsychological test scores, Rohling and colleagues (2002) failed to find a link between depression and objective neuropsychological functioning. These findings are similar to that of other researchers who attempted to investigate the effect of depression on cognitive functioning (Kaufman, A., Grossman, & Kaufman, N., 1994; Grossman, Kaufman, Mednitsky, Scharff, & Dennis, 1994; Miller, Faustman, Moses, & Csernansky, 1991).

Differences in study findings with respect to research investigating the impact of depression on cognitive functioning may be due to methodological differences (diagnostic inclusion criteria, etc.). Despite equivocal research findings regarding the effect of depression on neuropsychological functioning, there is a great deal of anecdotal support on the effect of depression on social, behavioral, emotional, vocational, and cognitive functioning (by self-report) in patients who sustained mTBI. Consequently, some mTBI sufferers do not return to work, are dependent on others, refrain from participating in pre-injury activities, experience increased family stress, demonstrate reduced ability to problem solve when faced with challenge, and are reported to have personality changes that affect how they interact with their environment

following the head injury (Curran, Ponsford, & Crowe, 2000). Moreover, the prolonged course of post-concussive symptoms may be construed by many clinicians as resulting from psychological and psychosocial factors, as opposed to physiological disturbance alone. Nevertheless, researchers suggest that psychological disturbances, such as depression, prolong post-concussive symptoms long after recovery of functioning was expected (Ruff, Camenzuli & Mueller, 1996; Yang, Tu, Hua, & Huang, 2007). Possibly, temporary cerebral dysfunctions and structural anomaly might have been the underlying factors of PCS initially; however, depression is purported to be responsible for persisting post-concussive symptoms (Machulda et al. 1998). As many of the symptoms of depression are common to post-concussive symptoms, it is reasonable to suggest that mTBI patients may continue to report symptoms and attribute these disturbances to the head injury sustained.

Current Treatment Strategies

In spite of the continuing controversy concerning the relationship between head injury and depression, the finding that depression is deleterious to perceived or actual recovery from concussive symptoms is consistent (Busch & Alpern, 1998; Varney & Roberts, 1999). Consequently, treatment for depression, regardless of origin, is important in this population. Promising results, including the increase in overall functioning (social, occupational, emotional) following treatment for depression, have been documented in mTBI and other neurologically impaired patients. Recommended methods to treat depression in neurologically impaired populations mostly include either pharmacologic treatment or psychotherapy. The former is likely the first line of treatment, as depression in the mTBI population is likely identified by a medical professional. However, long term pharmacologic treatment is expensive and its effectiveness has not been clearly established. Ragnarsson's (2006) review of goals that have gone unmet since 1998 include the lack of empirical data that substantiate the effectiveness of pharmacological interventions in treating emotional concerns. Psychotherapy, specifically cognitive-behavioral therapy, has been proven to be effective at treating depression (Beck, 1967; Nezu, 2000).

However, to date, no study has empirically validated the use of psychotherapy in treating depression following head injury. The following will briefly review research studies that have investigated current treatment strategies for depression following mTBI, including the reported effectiveness of these strategies.

Review of Pharmacological Treatment of Depression Secondary to mTBI

Depression is likely to be identified by a primary care provider or medical specialist (i.e., neurologist). Therefore, depressive symptoms are likely treated medically. In a non-head-injured population, treatment for depression is most often rendered by a primary care physician and not by a mental health provider and, as a result, the first line of treatment is typically pharmacotherapy. There is a growing literature investigating the effects of antidepressants and other psychotropic medications on depression in the mTBI group (Alderfer, Arciniegas, & Silver, 2005; Fann, Katon, & Uomoto, 2000; Fann, Uomoto, & Katon, 2001; Jorge, 2005; Kile, 2006). Specifically, the use of monoamine oxidase inhibitors (MAOI) and tricyclics resulted in inconsistent findings (Dinan & Mobayed, 1992; Saran, 1985; Saran, 1988; Tyler, McNeely, & Dick, 1980). More recent studies on the use of serotonin reuptake inhibitors offer evidence that psychotropic medications can be effective in treating depression secondary to head injury. Additionally, the findings of these studies support the notion that treatment of depression results in an increase in overall functioning.

Use of Tricyclic Antidepressants to Treat Depression following an mTBI

Saran (1985, 1988) investigated the effect of tricyclics on depression in a closed head-injured sample. Saran compared the mTBI group's response to a control group's response to amitriptyline, a tricyclic. The mTBI group was comprised of mildly head-injured individuals whose injuries were sustained less than one year prior to the start of the study; each of these individuals was diagnosed with major depression with melancholia in accordance with the Diagnostic and Statistical Manual of Mental Disorders III-R (DSM-III-R). Results from this study indicate that both groups began with comparable scores on baseline measures of

depression; however, only the non-head-injured participants benefited from treatment with the tricyclic (Saran, 1985). Similar findings were reported in a later study conducted by Saran (1988), wherein a mildly head-injured group with major depression was compared to a non head-injured group with major depression on the same psychotropic medications, amitriptyline and phenelzine. Kile (2006) cautioned against the use of tricyclic following mTBI, as the anticholinergic effects could exacerbate post-concussive symptoms, including any reported cognitive impairment.

Use of Monoamine Oxidase Inhibitor (MAOI) Antidepressants to Treat Depression following an mTBI

MAOI is reported to be effective in treating depression in patients who appear to be resistant to the effect of tricyclics. In the study conducted by Saran (1985) that investigated the effects of tricyclics in a mildly head-injured population, mTBI patients who had not responded to treatment with tricyclics were then administered an MAOI (Saran, 1985). However, the results of this study indicated that mTBI patients also failed to respond to the MAOI following four weeks of treatment, which would have been a long enough trial to yield effective results if any. Saran did find that the scores on anxiety and somatization indices of the HAM-D appeared to change. However, this change in score did not reach statistical significance (Saran, 1985).

Use of Selective Serotonin Reuptake Inhibitors (SSRIs) to Treat Depression Following an mTBI

Fann, Uuomoto, & Katon (1996) conducted a study that investigated the effect of SSRIs on the decrease of depressive symptoms, perceived distress, disability, and post-concussive symptoms, in individuals with major depression following an mTBI. These researchers used a within subject design wherein all subjects received placebos. Compared to the placebo trial, the mTBI patients experienced a greater decrease in scores on measures of depression, following SSRI use. Further findings suggest that not only did depressive and PCS symptoms decrease, but also physical and social functioning improved. Another study conducted by these researchers in 2001 resulted in similar findings (Fann, Uomoto, & Katon, 2001).

SSRIs do appear to be the most effective pharmacologic treatment for major depression following a mild head injury. Therefore if medications are included as an additional treatment modality to psychosocial/psychological interventions, SSRIs should be considered. The clinician is cautioned to monitor the side effects closely following the use of SSRIs, as these medications may exacerbate post-concussive symptoms if they are present. This effect may be counter-productive even if some reduction in depression is obtained, as side effects may result in an exacerbation in mood decline in the long-term (Kile, 2006).

The variation in effectiveness, from no improvement (tricyclics and MAOIs) to some improvement in depression (SSRIs) and the expense of ongoing medication use may be an obstacle in using medication as the primary treatment for depression in this substantially sized population (Alderfer, Arciniegas, & Silver, 2005). Prohibitive aspects of pharmacotherapy as the first line of treatment include the cost of medication use in the long-term, variability in effectiveness, and the risk that medication may exacerbate post-concussive symptoms (e.g., headache, dizziness, fatigue, nausea, decreased sexual functioning, et al.) (Kile, 2006).

It may be that increased sadness and hopelessness result from decreased ability to cope (resulting in negative cognitive schemas, behavioral inactivation, etc.), rather than biological (i.e., neurochemical or structural) factors that may be amenable to medication treatment. Therefore, pharmacotherapy may result in less benefit than cognitive-behavioral or other psychosocial interventions, even when neurovegetative symptoms associated with depression are improved. Additional support of a poor coping hypothesis may be observed through Saran's (1988) studies. Saran (1988) indicated that mTBI patients appeared to demonstrate psychiatric qualities and personality changes that might preclude effective treatment of depression. He stated that "exaggeration of hypochondrial concern, somatization, and conversion symptoms … preinjury neurotic tendencies, and personality changes" that affect coping response" (Saran, 1988, p. 81) likely contribute to persisting depression. Saran followed the patients in his study for 18 months and there was no objective pre-injury personality data collected. Therefore his assertions about

premorbid psychological functioning and its impact on patients' current symptom presentations are speculative. Nevertheless, his reported observations about his patients' responses strongly implicate mTBI patients' decreased ability to cope with the changes associated with their injuries. Improving one's ability to cope or adjust to changes associated with an mTBI is better addressed with psychological interventions than pharmacotherapy. Unlike the purpose of medication (symptom reduction), psychological interventions are designed to increase one's ability to adjust thinking patterns and behaviors.

Suggested Pharmacologic and Psychotherapeutic Treatments for Symptoms of Depression Based on Type of Depression

Pharmacological treatments, specifically the SSRIs, may be helpful in treating some symptoms associated with major depression (Alderfer, Arciniegas, & Silver, 2005; Fann, Uomoto, & Katon, 2001). However, further investigation is needed to replicate Fann and colleagues' (1996) findings. As suggested previously, psychotherapy is likely indicated to address poor coping, which is also associated with depression following mTBI. Onswerth and Oei (1998) suggest that multimodal treatment may be more effective at reducing depression secondary to head injury than unimodal treatment strategies (i.e., psychotherapy or pharmacotherapy alone). They further suggest specific treatment combinations for three basic categories that describe how depression may develop following an mTBI: 1) depression that develops within a brief time following the injury; 2) depressive syndromes that result from actual damage to brain structures; or 3) reactive depression that typically develops later in the recovery period from a head injury.

Early onset of depression following mTBI. Onswerth and Oei (1998) suggest cognitive-behavioral treatment for depression that develops early in the recovery process. These individuals may develop poor motivation or social withdrawal, which are often associated with the brain injury. These clinicians believe that psychotherapy, especially one that is goal directed and includes psychoeducation, could facilitate self-awareness and realistic self-appraisal. The use of pharmacotherapy for this group may be indicated if depression inhibits active participation in

their own rehabilitation. However, if not necessary to enhance activity in rehabilitative activities, it is suggested that the clinician not use medication as first-line treatment in this group. It is argued that medication may compromise cognitive functioning, precluding effective rehabilitation.

Depressive syndrome following damage to brain structures. It is possible that depressed mood may follow damage or compromise to neurological structures that are thought to be responsible for mood. It is likely that this type of depression is more responsive to pharmacotherapy than cognitive therapy models when compared to depression that results from perceived cognitive loss. However, Onswerth and Oei (1998) suggest that behavior modification techniques may be effective at shaping behaviors that were compromised by the injury. Cognitive therapy may be better indicated for individuals who have maintained higher levels of intellectual integrity than for those who have suffered more intellectually compromising injuries. Cognitive therapy, as opposed to just behavioral medication alone, may also be helpful to address depressed mood that results from poor coping with mTBI, following which intellect is likely still intact.

Reactive depression with later onset. Reactive depression likely develops later in the recovery process. It has been asserted that this type of depression is due to a poor coping response to the trauma associated with the actual injury or deficits that followed the head injury. Onswerth and Oei (1998) suggest that cognitive-behavioral techniques be used to treat these patients. These patients are likely to have a negative worldview following their injuries that spawns maladaptive thoughts, negative self-statements, and cognitive distortions (Beck, 1999). Onswerth and Oei (1998) report that the effectiveness of pharmacotherapy is questionable and not well investigated in this group.

Onswerth and Oei's suggestions on how to treat depression following head injury are the most comprehensive to date. However, their guidelines would require immense medical and psychological resources in addition to the medical care that head injury sufferers receive.

Moreover, their recommendations for treatment are rational, but are based on theoretical categories of depression and treatment recommendations that have not been empirically validated in a head-injured sample. Their recommendations do, however, highlight the importance of recognizing possible underlying etiology for psychological consequences for head injury. For example, it is presumed that patients who develop a large number of symptoms that increase in severity over time during the expected recovery period from a head injury are likely experiencing significant difficulties with coping with changes that have resulted from their injuries.

One treatment suggestion that remains constant in each of the depression categories is psychosocial education that assists the head-injured individuals in coping with their condition. Perhaps treatment models (similar to psychosocial education) used for post-concussive syndrome, a syndrome that likely shares similar substrates to depression following head injury, may also be helpful in reducing depression.

Chapter 4: Psychosocial Treatment for PCS as it Relates to Treating Depression Following mTBI

Psychosocial interventions are effective in decreasing the rate of PCS following head injury (Mittenberg, Canyock, Condit, & Patton, 2001). As identified earlier, it is unclear if the report of post-concussive symptoms is due to physiologic disturbance, poor adjustment to perceived or actual cognitive loss, or psychological disturbance (i.e. depression). However, mounting evidence suggests that poor coping ability and stress contribute to the persisting experience of PCS symptoms. As such, treatment strategies aimed at reducing the stress associated with perceived impairment and changes in environment following a head injury are being developed. For one such strategy, Mittenberg and colleagues (1994) developed a structured treatment manual for individuals with mild head injury. In this manual, the researchers included information that was found to be effective in reducing the anxiety that was related to poor understanding of head injury sequelae. They included information about head injury symptoms, including severity and course. Additionally, exercises that encouraged the cognitive reattribution of symptoms and cognitive behavioral treatment for emotional sequelae are included. Ponsford and colleagues (2002) had similar findings when they provided education about expected mTBI symptoms and suggested coping strategies. The participants who did not receive the intervention reported more post-concussive symptoms and greater distress than participants who received the intervention. Although not specifically targeting depressive symptoms, information about expectations and coping strategies to address expected problem were found to encourage affective coping skills that were aimed at precluding PCS development (Mittenberg et al., 1994; Ponsford et al., 2002). Perhaps targeting cognitive attribution and coping will also help prevent depression from developing following mTBI. These studies identified the use of interventions early in recovery to prevent threats to the recovery process. However, not much has been done to investigate the effectiveness of psychosocial interventions specifically on depression, which also develops following mTBI and is more relevant to the current study (Mittenberg et al., 1994).

Pennebaker (1986) developed an emotional disclosure paradigm, which may be an effective tool to develop affective coping skills following mTBI. To reduce depressive symptoms, emotional expression through writing about one's thoughts and feelings about one's head injury may be an alternative to structured treatment and educational programs that Mittenberg and colleagues (1994) and Ponsford and colleagues (2002) designed to promote development of affective coping skills to address head injury symptoms. Although the mechanisms that are responsible for the subsequent changes that result from utilizing emotional disclosure are not well known, what is known is that individuals who use it appear to cope with stress differently. The following will provide a basic review of the emotional disclosure paradigm and its use in clinical populations, including depressed samples.

Chapter 5: Written Emotional Expression: A Review

The use of written emotional expression (EE) has consistently resulted in a number of positive physical, psychological, and behavioral changes. Several studies report that writing or talking about an emotionally charged event results in higher grade point averages, decreased absenteeism from work, greater immune functioning, higher return to work ratio following termination of employment, decrease in anxiety and depression, as well as many other positive health outcomes and behaviors (Beckwith, Greenberg, & Gevirtz, 2005; Cameron & Nichols, 1996; Davison, & Thomas, 1995; Esterling, Antoni, Fletcher, Margulies, & Schneiderman, 1994; Francis & Pennebaker, 1992; Frattaroli, 2006; Frisina, Borod, & Lepore, 2004; Low, Stanton, & Danoff-Burg, 2006; Pasquale, Borad, & Lepore, 2004; Pennebaker & Beall, 1986; Pennebaker, Colder, & Sharp, 1990; Pennebaker & Francis, 1996; Petrie, Booth, Pennebaker, Spera, Buhrfeind, & Pennebaker, 1994; Smith, Kloss, Kniele, & Anderson, 2007).

The typical paradigm involves random assignment of participants to groups wherein they are instructed to write about what Pennebaker describes as their deepest thoughts and emotions associated with a traumatic event or an emotionally neutral event. Several measures are administered at baseline and then again at follow-up and experimental groups are compared to each other. Additionally, a measure of immediate mood state is administered prior to and following the disclosure. Since Pennebaker's landmark study in 1986 in which he investigated the effects of emotional disclosure on health center visits in college students, many researchers have conducted studies using the emotional disclosure paradigm in clinical populations (Beckwith, Greenberg, & Gevirtz, 2005; Frattaroli, 2006; Frisina, Borod, & Lepore, 2004; Kelley, Lumely, & Liesen, 1997; L'Abate, Boyce, Frazier, & Boyce, Low, Stanton, & Danoff-Burg, 2006; Maggio, 2007; 1992; H. Rosenberg, S. Rosenberg, Ernstoff, Wolford, Amdur, Elshamy, et al., 2002; Sandgren & McCaul, 2003; Stanton, Danoff-Burg, Sworowski, et al., 2002). Moreover, more recent emotional expression (EE) studies have investigated the effect of disclosure on psychological functioning.

Potential Mechanism of Change

As stated above, the mechanisms responsible for the changes observed following an emotional exercise are not clear. Pennebaker (2000) himself has evolved his theory as to why disclosure is effective. Initially, Pennebaker and Beall (1986) proposed that pent up negative emotion resulted in additive stress to the body. As a result, the body was vulnerable to illness and disease. By expressing the negative thoughts and emotions, Pennebaker (1986) initially asserted that this low level stress subsided, which resulted in better health and better mood in the long term. However, in response to studies that found no impact of disinhibition on the effect of EE, Pennebaker (1996) revised his theory. Additional studies that investigated this theory arrived at inconclusive findings or findings that did not support this initial theory (Consedine, Magai, & Bonanno, 2002; Kloss & Lisman, 2002; Greenberg, Wortman, & Stone, 1996).

Currently, he among other researchers posits that cognitive changes are necessary for the changes that occur as a result of disclosure (Consedine, Magai, & Bonanno, 2002; Donnelly & Murray, 1991; Pennebaker, 1997; Pennebaker & Frances, 1996; Kennedy-Moore & Watson, 2001). This proposed mechanism of change is vague; however, researchers have spent the greater part of the last decade clarifying the cognitive mechanisms that may be responsible for the changes that are observed following an emotional expression exercise. More specifically, several cognitive mechanisms are described by several researchers to be responsible for adaptive coping, including the development of cognitive schemas that assist in adaptive affective coping by extinguishing the distress associated with stress or trauma; the development of a story line that allows one to arrive to increased insight about the trauma; and/or the development of language that promotes cognitive organization of the trauma (Berry & Pennebaker 1993; Donnelly & Murray, 1991).

For example, emotional expression may allow the participants to self-regulate their emotions, as observed in both emotional expression studies and in psychotherapy, to facilitate both physical and mental health changes. Self-regulation may mediate negative affect and the

cognitive schemas created when disclosing about stressful or traumatic experiences (Cameron & Nichols, 1998). Cameron and Nichols (1998) proposed that writing facilitates adjustment to traumatic events by enabling the writer to develop a cognitive representation of the event that is, in essence, easier to cope with. More specifically, Cameron and Nichols (1998) investigated the effectiveness of a self-regulation task above and beyond the typical disclosure exercise. Their findings suggest that emotional self-regulation may be an important factor for individuals who have a negative attributional style (pessimists). The results suggested that emotional disclosure with and without a self-regulation task resulted in improved health, when compared to controls. They also found that mood state and college adjustment improved following disclosure and the self-regulation task in both experimental groups. However, pessimists benefited from the disclosure with the added self-regulation task and not the disclosure exercise alone. Possibly, pessimistic individuals may be accustomed to cognitive schemas that perpetuate negative patterns and negatively unbalanced cognitive representations of stressful events. Therefore, in the same way in which disclosure tasks assist individuals in developing balanced cognitive representations of their stressful or traumatic events, self-regulation tasks further assist pessimists in developing more balanced cognitive representations.

Pennebaker (1997) asserted that language and the organization of language over time result in better memory for the event, which results in improved active coping. He found empirical support for language as a possible mechanism contributing to changes observed following EE. He and colleagues researched the change in narrative use over study trials. Using a word analysis program, they found that, generally, positive outcomes were associated with specific types of language use. A link among language organization, the change in language use over time, and word type was revealed. These factors correlated with the therapeutic benefit in various populations who participated in emotional expression exercises (Pennebaker & King, 1999). Additional support for the impact of language organization and structure was observed in studies that found that instructing participants to cognitively reframe their trauma or focus on

perceived benefits from the trauma at the very start of their writing resulted in positive outcomes (King & Miner, 2000). (For review, please see Pennebaker**,** 1996). Schwartz & Drotar's (2004) findings also supported this, as they found a decrease in negative emotion words in the context of increased cognitive processing language positively impacted the scores on a physical health inventory.

Use of Emotional Expression to Decrease Depression

Two studies investigated the use of emotional expression in depressed populations. One such study by L'Abate, Boyce, Frazier, & Russ (1992) utilized a home writing design, in which all of the emotional expression exercises were completed in the participants' homes. L'Abate and colleagues (1992) conducted a series of studies to investigate the effects of writing on individuals who were depressed, as indicated by self-report on a measure of depression. Students who scored within the depressed ranges were recruited and randomly assigned to either a control group or one of three structured emotional expression groups, based on either Beck's (1974) cognitive model, L'Abate's (1986) interpersonal model of depression, or the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) Content Scale of Depression. Each student completed programmed writing exercises once weekly for ten weeks. The emotional expression groups resulted in decreases in the report of depression, irrespective of the severity of depression initially assessed at baseline and the type of structured writing used. However, these researchers did not include a long-term follow-up study, which precluded their making inferences about the effect of writing on depression in the long term.

Second, Lepore (1997) investigated the effectiveness of EE on depression in a group of college students who were scheduled to take graduate entrance exams. In contrast to number of trials that L'Abate and colleagues (1992) used for their study, Lepore investigated the effects of a one-time writing trial on depression. In this study, he interviewed students over the phone on three occasions to obtain descriptive information and depression scores. Ten days before the graduate exam, students wrote about their stress in the laboratory. Lepore found that participants

assigned to the emotional expression group demonstrated a decrease in scores on the depression measure, whereas the participants who wrote about trivial topics did not demonstrate any change. The author concluded that the depressive symptoms were reduced due to the attenuation of negative intrusive thoughts about an impending stressful event. In essence, he hypothesized that negative thought patterns were reduced. As these negative cognitive patterns may be responsible for the depressed mood that results from the individual's inability to cope with change following the mTBI, this manipulation may be particularly effective at reducing depression in this population.

Several studies objectively documented a decrease in depressive symptoms following emotional expression (Esterling, L'Abate, Murray, & Pennebaker, 2001; L'Abate, Boyce, Frazier, & Russ 1992; Lepore, 1997; Pennebaker, Mayne, & Francis, 1997). Therefore, the effectiveness of this paradigm in a depressed population has already been established. To date, few studies have used this model to investigate the effect of EE on psychological symptoms observed in medical populations. In one of few examples of this application, Stanton and colleagues (2002) investigated the effects of EE on affective and psychological adjustment to breast cancer. They found that EE was effective at improving adjustment and decreasing distress. However, this effect was attenuated by one's level of avoidance. Nevertheless, this study did not specifically study EE's effectiveness on depression in this medical population, which is the focus of the current study.

It has been presented that poor understanding of symptom course and perceived decrease in functioning likely result in increased distress, decreased coping, and negative cognitive patterns, which may be a factor in the development of depression following head injury. Therefore, emotional expression may assist depressed, head-injured patients in creating organized, cognitive representations of their situations as a result of their head injuries. This may result in improved active coping, which may facilitate a decrease in depression. Moreover, research supports that it may be necessary to include a self-regulation task with an EE exercise

that is designed to reduce depression. Without intervention, depressed individuals likely reconstruct their stories using negative cognitive schema and patterns. More specific to the current research proposal, depression that develops following head injury may have developed following poor affective coping. Self-regulation as an addition to an emotional expression exercise may be key in mediating negative affect when reconstructing a narrative that is reflective of improved psychological outcome. Therefore, such a task might prove effective in reducing depression in an mTBI population.

There are two additional factors that may be important to the design of the proposed study after considering each of these studies that investigated EE and its effect on depression. The first is that L'Abate (1997) and colleagues utilized an unprecedented extended writing model that required the participants to write over ten separate occasions. On the other hand, Lepore (1997) required participants to write on one occasion only. Each of these studies resulted in decreases in scores on depression measures. The majority of EE studies, however, require participants to write over three to four trials and physical health and behavioral changes are still evident. As the current proposed study will closely mirror the traditional emotional expression paradigm (three to four trials), it may result in additional information about whether three to four writing trials can result in a decrease in depression. The second noteworthy point is that studies that investigated the effect of emotional expression on depression utilized home writing designs. Kelly, Lumely, and Leisen (1997) also included a home-bound methodology when they investigated the effects of verbal emotional expression on rheumatoid arthritic patients. In this medical population, these researchers found that EE, though completed at home and not in research laboratory, resulted in improved mood and physical functioning. One disadvantage may be the lack of evidence that supports any long-term effects that EE exercises may have on depressed mood, as reported in research studies by L'Abate et al. (1996, 1997). Nevertheless, effects on depression in the short-term were found.

Malec (1999) asserted that cost-effective, unobtrusive interventions are necessary to develop to address emotional disturbance observed following mTBI. To date, no current studies purport any issues or concerns with ethics or adverse events as a result of using this format. Therefore, the goal of the present study was to develop a method whereby individuals who have suffered mTBI may experience a decrease in depressive symptoms, without further burden to the medical system through direct provider contact. A home-writing paradigm appeared to be an appropriate way to investigate this intervention in this population.

Chapter 6: Emotional Expression as a Treatment for Depression following mTBI

Depression following mild head injury may initially result from physiologic disturbance to the brain; however, as many sequelae are expected to subside in most patients within three months post-injury, so should any resulting depression symptoms (Busch & Alpern, 1998). With depression persisting for years post-injury in a significant percentage of mTBI patients, this is not the case. Therefore, many have subscribed to the notion that depression likely results from poor coping with environmental stress and the more immediate neurobehavioral changes that result immediately following a head injury, as opposed to persisting symptoms that are organically mediated (Busch & Alpern, 1998; Onsworth & Oei, 1998; McAllister , Saykin, & Flashman, 1999). Furthermore, empirical research supports this hypothesis. In the same way in which information given to a mTBI patient immediately following a head injury may assist the patient in coping with the injury, resulting in a decrease in PCS, EE may assist depressed patients with creating organized, cognitive representations of the effects of their injuries on their present condition to assist in coping. To date, no evidence has been found to suggest that the traditional EE paradigm exacerbates depressed mood in the long term. It is well documented that negative mood emerges immediately following EE in most populations, healthy or clinical. However, negative mood has been documented to decrease from trial to trial, resulting in an overall decrease in negative affect from pre- to post EE (Kloss & Lisman, 2001; Pennebaker, 1986).

In accordance with the findings of Curran and colleagues (2000) and the assertions by Kennedy-Moore and Watson (2001), restoring the locus of control for the mTBI patient may also assist in developing a coping strategy. Additionally, understanding expected disease course may pre-empt the development of a negative belief system that underlies negative cognitive patterns that may contribute to depressed mood. An EE exercise may be powerful in restoring the head-injured individual's ability to develop an active coping strategy, by allowing the individual with mTBI to potentially identify and confront negative thought patterns. Although specific psychological mechanisms are not well understood, disclosure likely assist individuals in

developing coherent, balanced cognitive representations of traumas or stressors, which remains within their control. This new schematic representation is believed to provide a cohesive, better understood storyline (with beginning, middle, and end) that may result in better coping due to a more balanced view of the injuries and the effects of these injuries. These cognitive schemas are then reflected in more logical story lines that may be reflected in the change in writing.

As emotional expression has been found to result in increased insight and organization, this model may also result in a decrease in emotional disturbance following mTBI. Therefore, the purpose of this study was to investigate the effect of emotional expression on individuals who experience depressed mood following mTBI at least six months post-injury. However, this study was also intended to test a format that will pose less burden on the health system. To accomplish this, interventions were employed via mail and phone.

Chapter 7: Rationale for Home-based Design with Structured Telephonic Support

A growing literature has documented the effectiveness of telephonic treatment (Barry, 2000; Bleich, 1998; Martin, Coyle, Warden, & Salazar, 2003; Morrison, Bergauer, Jaques, et al. 2001; Salazar, Warden, Schwab, Spector, Braverman, Walter, et al., 2000). More specifically, several studies investigated these techniques in traumatic brain injured populations. In these studies, researchers found that mailing materials and following up over the phone resulted in similar benefits to services provided in a clinical setting, which significantly adds to cost. For instance, Salazar and colleagues (1997) found that in moderate to severe TBI patients, structured cognitive rehabilitation conducted and prompted by phone resulted in similar benefit to cognitive rehabilitation conducted in their clinic. Moreover, also applicable to the procedures of this study, mail-in and telephonic procedures were used in the two disclosure studies that investigated the use of emotional expression in depressed populations (Lepore, 1997; L'Abate et al., 1997). As mentioned previously, a literature review addressing home-based designs for use with mTBI or depressed populations revealed no information about ethical or clinical problems related to this method. Additionally, concerns about utilizing this design for use with the emotional disclosure paradigm were not found. Particularly in the studies conducted by Lepore (1997) and L'Abate (1997) participants completed writing exercises that were mailed to them. Baseline and follow-up measures were administered completely over the phone in one of these studies, while the other administered all protocols through the mail. In each of these studies, writing exercises resulted in decreases in depression (L'Abate, 1997; Lepore, 1997).

Chapter 8: Research Study

The current study seeks to investigate the use of an emotional expression exercise to decrease depressed mood following history of mTBI, utilizing methods modeled after telephonic medicine (mail-in home-based designs). The following will outline the study's hypotheses, methods, and results. Lastly, the implications of this study's findings will be reviewed in the discussion.

Hypotheses

Hypothesis 1

Participants with mTBI who are assigned to the emotional expression group will demonstrate a decrease in their scores on the measure of depression, while no change will be observed on the scores of the mTBI participants assigned to the neutral writing group.

Hypothesis 2a

The emotional expression and neutral writing groups will demonstrate equal levels of negative affect (NA) and positive affect (PA), prior to beginning the first writing trial (pre-trial).

Hypothesis 2b

The experimental writing group will demonstrate a greater increase in NA from pre- to post-writing (immediately following each writing exercise), when compared to the neutral group.

Hypothesis 2c

The experimental group will demonstrate a decrease in NA from trial to trial (trial one to trial two, from trial two to trial three, etc.), while the control group will demonstrate no change in NA.

Hypothesis 2d

The experimental group will demonstrate an increase in PA from trial one to the completion of the three writing trials, while the control group will demonstrate no change in PA.

Hypothesis 3

A positive correlation between scores of depressive symptoms and PCS will be observed. For example, any decrease in depressive symptoms observed following emotional expression exercises will correspond to a decrease in scores obtained on the post-concussive symptom checklist. This hypothesis is based on the findings that depression and PCS are inter-related. Therefore, any change in symptoms associated with depression may influence the person's subjective report of PCS symptoms.

Hypothesis 4

High scores on the depression index will positively correlate with the behavioral disengagement, self-blame, self-distraction, and denial scales, while low scores will correlate with emotional support, instrumental support, positive reframing, planning, humor, and acceptance scales, as measured by the Brief Cope. It is well documented that depression results in decreased coping ability (Nezu, 2001). As such, the higher one scores on a depression measure, the more likely it is that one will endorse using the above maladaptive coping techniques to handle difficulties. The lower one scores, the greater the likelihood of using adaptive coping strategies.

Hypothesis 5

Participants assigned to the written emotional expression group will demonstrate higher scores on the scales that measure active coping, use of emotional support, instrumental support, positive reframing, planning, humor, and acceptance coping strategies, as measured by the Brief Cope, than participants assigned to the neutral writing group.

Method

Participants

Forty-two participants were recruited from the Defense and Veterans Brain Injury Clinic (DVBIC) of the United States Air Force's Wilford Hall Medical Center (WHMC). Each of these patients was a medical beneficiary of the Department of Defense (DoD), most of whom were Active Duty (AD) military members (98%). Organically mediated symptoms associated with

mTBIs are expected to subside within three months (spontaneous recovery). Therefore, only participants who reported sustaining a mild head injury at least six months prior to being recruited for this study were invited to participate in this study. Participants' head injury history was confirmed through a review of their medical records. Participants who endorsed experiencing depressed mood during the recruiting process, but declined participation in the study, were offered information about further evaluation and treatment services available to them at the Wilford Hall Medical Center or at their closest military treatment facility where military/DoD beneficiaries are eligible to receive medical services without cost.

Each participant met the following inclusion criteria: (a) sustained at least one reported mild head injury, as outlined by the mild Traumatic Brain Injury Subcommittee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation as outlined earlier (1993) (by self-report or as documented in medical records); (b) sustained the last head injury at least six months prior to beginning this study; and (c) reported having felt sad or anhedonic for at least two weeks prior to being recruited for the current study. Participants were carefully screened prior to being recruited and were also screened during the initial phone contact. Potential participants were not enrolled in the study if they met any of the following exclusion criteria during the review of the medical record or during the initial phone interview: (a) sustained hemorrhagic injuries as a result of the head injury; (b) reported focused and frequent suicidal ideation (e.g., daily thoughts of wanting to kill oneself that last more than one hour even though no plan is reported) or have a history of suicidal attempts or chronic hopelessness (by self-report); (c) previously diagnosed with a thought disorder, bipolar spectrum disorder, dementia, or any other severe psychiatric disorder; (d) previously diagnosed with a pervasive developmental disorder or any learning disorder that had not been treated; (e) reported current substance abuse (alcohol, illicit, or prescription); (f) reported deficiencies in speaking and/or writing English fluently; (g) sustained skull fractures or severe head injuries in past; (h) had significant changes in medications; (i) spent significant time in the hospital post-injury (greater than 30 days for mTBI

alone/ greater than 60 days for orthopedic injuries); and (j) involved in litigation due to injuries. Members who reported receiving psychiatric care that included medication management were included in this study. However, members who reported current psychological treatment (psychotherapy) for current depression and/or anxiety were excluded, as psychological treatment for anxiety and depression might confound the results of the current study.

There is concern about the tendency to exaggerate symptoms to obtain secondary gain in the mTBI population. As such, participants in litigation due to injuries were excluded. The majority of the participants were military service members. Due to Feres doctrine, military members are barred from suing the federal government. The one civilian participant was not involved in any litigation. Once the exclusion and inclusion criteria were assessed through a telephonic interview (self-report) and available medical documentation (medical records) was reviewed, all information was documented on the Descriptive Information Form (see Appendix E) and on the Medical Record Checklist (see Appendix I).

Demographic and Descriptive Information of Participants. Of the 42 participants recruited, 95% were male, while 5% were female. When asked about ethnicity, 57% described themselves as Caucasian, 24% as Hispanic, 12% as African American, and 7% as Asian American. All participants indicated that they spoke and wrote English fluently. Mean years of age was 28 years old (*SD*=4.9). The youngest participant was 21 and the eldest was 39. Mean years of education was 13 years (*SD* =1.3). The fewest years of education obtained was reported to be a high school diploma (12 years) and the greatest amount of education obtained was reported to be a 4-year degree (16 years). Several Chi-square tests were conducted for nominal level data and t-tests were conducted for parametric level data to determine if there were demographic differences between the experimental and control groups. No differences were found between these two groups when they were compared on gender $X^2(1, N = 42) = 1.29$, $p > .05$**,** ethnicity $X^2(3, N = 42) = .87$, $p > .05$**,** age $t\ (42) = 2.48$, $p > .05$**,** and education $t\ (42) = .760$, $p > .05$ **.**

Injury Profiles of Participants. The injury profile of participants was determined by: a) calculating frequencies for injury types; b) obtaining percentages of participants who sustained loss of consciousness (LOC) or who suffered post-traumatic amnesia (PTA); and c) calculating means for LOC and PTA time. Information about injuries was obtained from participants in telephonic interviews, which were cross-referenced with patients' medical records. Participants reported sustaining their mTBIs through improvised explosive devices (IEDs) (67%), combat blast injuries (12%), motor vehicle accidents (MVAs) (12%), vehicular-borne improvised explosive devices (VBIEDs) (7%), and falls (2%). With respect to the frequency of LOC experienced by the current sample, at the time of their injuries, 78.6% of the participants self-reported or were observed to have experienced an LOC, 9.5% reported feeling "dazed", while 12% denied having experienced LOC, but had experienced some form of post-traumatic memory loss. When frequency of PTA experienced by participants was calculated, it was revealed that 36% experienced some memory loss, while the remaining 74% denied having experienced PTA. The mean times of LOC and PTA were 3.9 minutes and 3.2 minutes, respectively. PTA time encompassed any period of LOC. Therefore PTA was expected to have a higher average than LOC. Further analysis revealed that several members either denied experiencing PTA or had missing values, which resulted in different sample sizes (n) for LOC and PTA. All participants denied having had a skull fracture or history of severe head injury, which was confirmed through reviewing participants' medical records. Participants reported experiencing a variety of orthopedic or non-neurological injuries including burns, fractures, embedded shrapnel wounds, and lacerations. The mean time spent in inpatient care was 33 days (*SD*=9.2). Experimental groups were compared on their injury profiles to determine any differences between the groups on injury type, experience of LOC/PTA, and/or LOC/PTA time. Chi-square tests were conducted to analyze nominal level data and t-tests were conducted to analyze parametric data. A t-test revealed a significant difference between groups on the amount of time reported for LOC; $t\ (42) = 2.17$, $p < .05$**.** The neutral group reported longer mean time for LOCs. No other significant

differences were found for injury type $X^2(5, N = 42) = 3.48, p > .05$, incidence of LOC (3x2) $X^2(3, N = 42) = 1.25, p > .05$, incidence of PTA, or PTA time $X^2(1, N = 42) = .48, p > .05$ and $t(42) = -.186, p > .05$, respectively. As participants were randomly selected and randomly assigned to each group, it is unclear the reason LOC times statistically differed between the two groups. This is possibly a random finding due to the small sample size.

Measures

Participants completed the following screening, process, and outcome measures, each of which are described below.

Screening measures.

1. Medical Record checklist: The associate principal investigator (AI) completed this checklist while reviewing the medical record. This checklist included all inclusion and exclusion criteria to determine patients' appropriateness to participate in the current study. The associate principal investigator documented the relevant information obtained from the medical record. However, data not available through the medical record were obtained through self-report during the telephonic interview. (Appendix I)

2. Descriptive Information: Participants completed brief questions about personal, academic, and medical information, including age, gender, level of education, how the head injury was obtained, time since injury, and severity of injury. This information was used to investigate any potential confounding variables that could impact proposed analyses (Appendix E).

3. DSM-IV-TR checklist for Major Depressive Disorder (MDD): This checklist included a list of symptoms taken from the DSM-IV-TR (2000) that indicated the likelihood of MDD. Participants were asked to endorse whether they had experienced each of the listed symptoms. Inclusion in the study was based on whether the individual experienced depressed mood prior to inclusion in the current study. To determine this, participants indicated that they experienced either depressed mood (or has been observed by others to demonstrate depressed mood) or

decreased interest in pleasurable activities for at least two weeks prior to the start of the study. In addition to documenting participants' report of depressed mood following their head injuries, this checklist also yielded additional information regarding whether participants met criteria for a major depressive episode at the time of participation in this study (Appendix D).

Baseline/outcome measures.

1. Post mTBI Symptom Checklist (Cicerone & Kalmar, 1995): This inventory is comprised of 30 items that described symptoms that are commonly experienced following an mTBI. Participants were asked to endorse items they had experience since sustaining their head injuries. Participants rated symptom severity, using a Likert scale, ranging from 0= "None" to 4 = "Severe." Therefore, the higher the score obtained, the greater the number and severity of symptoms experienced. This checklist documented potential presence and severity of post-concussive syndrome. The scores obtained on the measure have been previously found to strongly correlate with disability severity. (Appendix J).

2. The Brief Cope: (Carver, Scheier, & Weintraub, 1989). This measure is an abridged version of the Cope Inventory. Participants completed this 28-item measure that assessed coping style. Each item was endorsed on a Likert scale, ranging from 1, "I haven't been doing this at all" to 5, "I have been doing this a lot." Items were calculated into 14 separate indices. The coping indices of the Brief Cope include active coping, planning, acceptance, humor, positive reframe, religion, emotional support, instrumental support, self-distraction, denial, venting, substance use, behavioral disengagement, and self-blame. These indices have obtained alpha coefficient reliabilities between .50 and .90, which is supportive of internal reliability of the abbreviated scales. This measure was used as an outcome measure to investigate the impact of writing on coping style.

3. Neurobehavioral Functional Inventory (NFI): (Kreutzer, Seel, & Gourley, 2001). This inventory is comprised of 105 items that were grouped into six indices: depression, somatic complaints, memory/attention difficulties, communication deficits, aggressive behaviors, and

motor impairment. These items were rated on a four-point Likert scale, based on frequency- 1) never, 2) sometimes, 3) often, or 4) always. Cronbach's alpha for each scale ranges from .86 to .95. This measure was normed in a head-injured population. It was found to be highly correlated with related scales of the Minnesota Multiphasic Personality Inventory-II (e.g., Scale 2). This measure removes the physiological bias observed on other depression checklists and inventories that are often artificially elevated in patients with history of head injures due to symptom overlap. As such, it is reported to be more reliable at measuring symptoms associated with depression in a head-injured population. This measure was used as the primary outcome measure. Using this measure, depression severity was determined at baseline, at the conclusion of the last writing trial, and at the completion of the three writing trials. The purpose of this measure was to document the effect of EE on depressed mood. (Appendix K)

Process measures.

1. Positive and Negative Affect Schedule (PANAS): (Watson, Clark & Carey, 1988). This measure is a 20-item inventory that is a brief, sensitive measure of an individual's current affect. It is reported to be highly sensitive to changes in mood over brief periods of time, due to its high discriminant validity. Additionally, this measure has obtained alpha coefficient reliabilities between .84 and .90. Participants completed this short measure before and after writing to document transient positive and negative mood states due to writing (Appendix F).

2. Post-writing Questionnaire: Participants used a Likert scale to rate their writings on several questions that documented their reactions to and perceptions of the writing exercises. The purpose of this was to monitor the effect of writings on affect, as it identified whether the writing exercises elicited different responses from the experimental group when compared to the control group. These questions were adapted from previous disclosure studies (Appendix G).

Design and Procedures

The associate principal investigator (AI) reviewed the medical records of DVBIC patients who sustained mild traumatic brain injuries. Patients who sustained mTBI at least six

months prior to being recruited and who showed no evidence of meeting any exclusion criterion were deemed appropriate to recruit for this study. Upon the record review, the Medical Record Checklist was completed during which time the AI documented information pertaining to the exclusion and inclusion criteria. Each potential participant was contacted by phone. At the time of this initial call, the potential participant was screened and inclusion and exclusion criteria were reviewed for a second time and confirmed. The DSM-IV-TR (2000) Checklist for Major Depressive Disorder (MDD) was administered by phone to determine if the participant reported demonstrating symptoms consistent with MDD. If participants endorsed depressed mood, had sustained an mTBI, and was six months post-injury and did not endorse any of the exclusion criteria, as determined during the screening process, they were asked to participate in the current research study. Once participants verbally agreed, informed consents were verbally reviewed and participants were informed of the procedures they would be expected to accomplish, including options to decline participation in the study at any time. The informed consent, along with study materials were mailed to participants within two to three days following initial phone contacts.

As outlined in this study's proposal, participants were contacted within three days following the mailing of study materials to determine if they had received the study packet. Once participants received the study packet, they were then provided detailed information on how to complete the written informed consent, Descriptive Information form, Neurobehavioral Functional Inventory, and mTBI Symptom Checklist. Participants were encouraged to ask questions at that time. Following completion of these forms and questionnaires, participants were asked to include these materials in one of the addressed stamped envelopes provided, which were included in the research packets. Participants were asked to separately mail the informed consent back immediately in one of the stamped envelopes that were provided. It is important to note that this procedure was modified from initial procedures, in which participants were asked to mail the informed consent to the AI along with all of the other research materials. It was important to deviate from the initial proposal to ensure that all informed consents were completed properly and

returned to the AI in a timely manner. Following completion of the informed consent, Descriptive Information form, Neurobehavioral Functional Inventory, and mTBI Symptom Checklist, participants were prompted to begin the first writing exercise, as outlined in the research packets. Each participant was called on the two consecutive days following the first writing trial as a reminder to complete subsequent writing exercises on those days. The goal was to speak to participants directly at the time of these reminder calls. However, if any participant was not reached directly, a voicemail message was left for that participant to complete the appropriate writing trial that day. The AI's contact number was also left on the voicemail in case the participant had questions or if the participant wanted to report a significant change in status. During reminder calls, participants were asked if any urgent concerns arose since their last writing trial. No concerns were reported. Participants were also provided brief reminders regarding emergency procedures should emotional distress emerge that did not subside. After 20 minutes elapsed from the time participants began each of their writing trials, they were called again. At that time, participants were prompted to stop writing and to enclose their writings in the appropriate envelopes provided. Six weeks following the last writing trial, participants were mailed follow-up questionnaires (DSM-IV TR Checklist for Major Depressive Disorder, Descriptive Information form, Neurobehavioral Functional Inventory, mTBI Symptom Checklist, The Brief Cope, and Post-writing Follow-up questionnaire). The AI attempted to contact each participant via phone to assist in completing the six-week follow-up questionnaires. At the conclusion of the study, participants were debriefed about the study's purpose and findings.

Procedures for Writing Exercises. At the time of mailing, participants were randomly assigned to one of two groups, an emotional expression (EE) group or a neutral (N) writing group. Participants assigned to the emotional expression group were asked to write about any trauma related to their head injuries. Writing instructions, which were adapted from Pennebaker's (1986) previous disclosure exercises, were modified for the purposes of the current study and were provided to each participant and read by the AI over the phone prior to beginning each

writing trial. The experimental writing instructions included a self-regulation prompt, "Please include in your writing some things that you can do that may help you deal with the problems you have had." Prompts for the second and third writing trials included: "Have you tried the things that you thought could be helpful to deal with your problems? If so, were they helpful?" These prompts are modifications of the instructions used in the self-regulation task investigated by Cameron and Nichols (1998). According to research from Cameron and Nichols (1998) this prompt may be effective at balancing negative cognitive representations and promote emotional adjustment in previous studies (Cameron & Nichols, 1998; King & Miner, 2000). (See Appendix A for complete writing instructions.) Participants assigned to the neutral group were asked to write about what they had accomplished that day from the time they had awakened. Participants of the neutral group were asked to document only the facts of that day and were instructed to keep their writings free of emotions (detailed thoughts about how they felt about their day and any feelings in general).

Participants completed the PANAS, a measure of positive and negative affect, before and after completing each writing exercise. Following the first day's procedures, participants were educated on the additional two packets that contained materials for two additional writing exercises, which were accomplished over the next two consecutive days following the first writing trial. Six weeks following the initial writing, participants were mailed follow-up questionnaires and were called by the AI to initiate/assist with questionnaire completion. When difficulty arose in obtaining information through the mail, participants were contacted via phone. If the follow-up questionnaires were not received from participants, all attempts to collect data were frequently attempted by phone, mail, and electronic mail.

Analysis Strategy

A priori power analysis was conducted to determine the number of participants that were needed for this study. To test the difference between means of the outcome data that would be obtained from the two groups, it was projected that, based on d=7 (medium to large effect size),

the power of .80, and alpha level set at .05 for t-tests and *f*=.2 (medium effect size), the power of .75, and alpha level set at .05 for repeated measures ANOVA, 52 participants would be needed to meet sufficient power to achieve results for this study. However, following rigorous recruitment over a protracted period, only 42 patients agreed to participate in this study. Despite concentrated efforts to collect data via phone, mail, and electronic mail on multiple occasions, only 13 participants returned the all three writing trials of the study along with their informed consents. Only one participant returned his six-week follow-up data. The data obtained were checked for integrity (missing data were coded consistently and minimum and maximum scores were calculated to assure that scores were within expected ranges). SPSS was used to conduct all statistical analyses.

It is likely that the low number of participants included in the following analyses precluded the data from reaching the amount of power necessary to detect significant differences between the EE and N groups on the process and outcome measures, utilizing a .05 cut-off for the p value. However, this statistical problem also resulted in an inability to reject the null hypotheses (Cohen, 1994). Therefore, additional analyses were utilized to analyze data.

Despite the low number of participants, emotional expression and neutral groups were compared on descriptive information, demographics, and mTBI symptoms to ensure equality of groups and to document if there were any potential confounding factors. Chi-square tests were conducted on nominal data (e.g., gender, education level, injury type, etc.), while t-tests were employed to investigate parametric data (e.g., age, time since injury, etc.). Those who completed the study were compared to those who did not complete the study on demographic and descriptive data obtained through medical records and self-report. Lastly, post-hoc analyses were conducted to provide an in-depth view of the data that were obtained, as it appeared that the inability to reach the necessary power prevented the proposed statistical procedures from yielding useful information. Therefore, those who appeared to benefit from the writing trials (responders) were compared to those who did not appear to benefit (non-responders), based on changes in

depression scores. Additionally a qualitative review of participants' narratives was also accomplished to determine any perceivable writing differences between the responders (those who demonstrated any change on their depression scores) and non-responders (those who demonstrated no change on their depression scores). The implications of these findings are speculative at best. However, this information may yield a better understanding of factors to consider when developing future studies that investigate ways to manage depression in this population.

Chapter 9: Results

Of the 42 participants, 13 (n = 13) completed and returned a portion of the research study, as outlined in the proposal. Only these individuals were included in the overall analyses. One participant (Neutral group participant) completed the entire study, including the questionnaires included in the six-week follow-up trial. The demographic information of participants who returned at least the first three trials of study materials (completers) were compared to participants who had not returned their study materials (non-completers). Chi-square tests and t-tests were conducted and no differences in gender $X^2(1, N = 42) = .84$, $p > .05$, ethnicity $X^2(3, N = 42) = 3.16$, $p > .03$, age $t(42) =$ -.721, $p > .05$**,** or education $t(42) = -.215$, $p > .05$ were found between completers and non-completers. Additionally, no differences in most factors on the injury profile emerged, as the groups appeared to be similar in injury type $X^2(5, N = 42) = .17.75$, $p > .05$, incidence of PTA $X^2(1, N = 42) = 2.67$, $p > .05$, and time of LOC $t(42) = -.368$, $p > .05$. However, counter to expectation, the groups did differ on PTA time and incidence of LOC $t(42) = -2.13$, $p = .002$ and $X^2(3, N = 42) = 8.73$, $p = .03$, respectively**.** Further review showed that the completers had higher mean PTA time. 92% reported sustaining their injuries from IED blasts, versus the various other injury types that the non-completers reported. The following analyses were conducted using the 12 participants who completed at least the first three trials.

Hypotheses, Aims and Findings

Hypothesis 1

Participants with mTBI who are assigned to the emotional expression group will demonstrate a decrease in their scores on the measure of depression, while no change will be observed on the scores of the mTBI participants assigned to the neutral writing group.

Aim 1. Determine the effect of an emotional expression exercise on depression in an mTBI sample, compared to a neutral writing exercise, as measured by a self-report measure of depression, the Neurobehavioral Functioning Inventory (NFI).

Participants in the N group were compared to participants in the EE group on scores

obtained on the Depression scale of the NFI. No differences in depression scores were evident at baseline. A repeated measures ANOVA revealed no significant difference between the two groups on the Depression scale of NFI ($F(1, 12) = .056$, $p > .05$), following the three writing trials. T-tests were conducted to compare the means of initial scores and then the means of scores obtained after the writing trials. Neither of these revealed significant differences between the two groups $t(12) = .50$, $p > .05$ and $t(12) = .43$, $p > .05$, respectively. Due to the low number of participants, data were plotted on a chart to determine if a directional trend could be detected when the groups were compared to each other. This depicts the change in the mean scores for the two groups, with the EE group dropping approximately one-point from baseline to following the three writing trials, while the N group essentially remained the same from baseline to the end of the writing trials (see graph in Appendix L).

Hypothesis 2a

The emotional expression and neutral writing groups will demonstrate equal levels of negative affect (NA) and positive affect (PA), prior to beginning the first writing trial (pre-trial).

Hypothesis 2b

The experimental writing group will demonstrate a greater increase in NA from pre- to post-writing (immediately following each writing exercise), when compared to the neutral group.

Hypothesis 2c

The experimental group will demonstrate a decrease in NA from trial to trial (trial one to trial two, from trial two to trial three, etc), while the control group will demonstrate no change in NA.

Hypothesis 2d

The experimental group will demonstrate an increase in PA from trial one to follow-up, while the control group will demonstrate no change in PA.

Aim 2. Determine the effect of the emotional (experimental) and neutral writing exercises on immediate positive and negative affect (PA and NA, respectively) in an mTBI population.

Repeated measures ANOVAs were conducted for both positive and negative affect scores that were obtained prior to and following each writing trial. As expected, no significant differences were found between the groups prior to the first trial. In fact, no significant differences were found to support any of the hypotheses projected for Aim 2, 2a.-2d. As significant findings were not expected due to small sample size, an additional method to that hypothesized was used to analyze these data. The means of the two groups were closely examined to determine if any trends were observed when the groups were compared to each other on changes in affect following each writing trial. As expected, the EE and N groups possibly demonstrated different patterns of change in affect when the groups were compared to each other. Regarding negative affect, as projected, the EE group demonstrated an increase in negative feelings, while the N group demonstrated a decrease in negative feelings, following the first writing trial. This trend continued for the EE and N groups following the second writing trials. Following the last writing trial, the EE group continued to show an increase in negative affect in response to their writings, while the N group demonstrated little or no changes. With regard to positive affect, against expectations, the EE group demonstrated decreases in positive affect following the first and second writing trials. However, as projected, the EE group demonstrated increases in positive affect following the last trial. The N groups also appeared to respond similarly to projections for the first and last trials, as little change in positive affect was noted. The exception to this was the decrease in positive affect following the second writing trial, which was not expected by the N group. Lastly, as hypothesized, the EE group appeared to demonstrate a decrease in the level of negative affect in pre-trial scores from trial 1 to trial 2. This change was not observed from trial 2 to trial 3. However, trial 3 negative affect scores still remained lower than what was observed on trial 1 prior to the EE writing exercise (see graphs depicting negative affect for N and EE groups in Appendix L and Appendix M).

Hypothesis 3

A positive correlation between scores of depression and PCS will be observed. For instance, any decrease in depressive symptoms observed following emotional expression exercises will correspond to a decrease in scores obtained on the post-concussive symptom checklist. This hypothesis is based on the findings that depression and PCS are inter-related. Therefore, any change in depression may impact the person's subjective report of PCS symptoms.

Aim 3. Determine the relationship between depression and post-concussive symptoms in a depressed head-injured sample from baseline to the completion of writing the three trials.

Pearson correlations were conducted to determine any relationships between depression scores and PCS symptoms. Baseline depression and PCS symptoms were positively correlated $r(12) = .832$, $p = .001$. Therefore, as depression scores increased, PCS scores also appeared to increase. No relationship between these two variables were found following the three writing trials, $r(12) = 465$, $p > .05$.

Hypothesis 4

High scores on the depression index will positively correlate with the behavioral disengagement, self-blame, self-distraction, and denial scales obtained on the Brief Cope, while low scores will correlate with emotional support, instrumental support, positive reframing, planning, humor, and acceptance scales. It is well documented that depression results in decreased coping ability (Nezu, 2001). As such, the higher one scores on a depression measure, the more likely it is that one will endorse using the above maladaptive coping techniques to handle difficulties. The lower one scores, the greater the likelihood of using adaptive coping strategies.

Aim 4. Identify the relationship between depression, as measured by the Neurobehavioral Functional Inventory (NFI), and coping ability, as measured by the Brief Cope in individuals who have sustained mTBI.

Pearson correlations were conducted to determine potential relationships between

depression and maladaptive coping strategies, behavioral disengagement, self-blame, self-distraction, substance abuse, and denial scales of the Brief Cope. As hypothesized, a statistically significant correlation was found between depression scores and the behavioral disengagement coping index when baseline scores were analyzed, $r(12) = .697$, $p = .012$. In essence, as depression scores increased, scores on the behavioral disengagement index also increased. No other significant relationships were found when baseline scores were analyzed. Correlations between coping and depression scores were compared again, following the three writing trials. The correlation between depression scores and the behavioral disengagement index was no longer significant, $r(12) = .067$, $p > .05$.

Hypothesis 5

Participants assigned to the written emotional expression group will demonstrate higher scores on the scales that measure active coping, use of emotional support, instrumental support, positive reframing, planning, humor, and acceptance coping strategies, as measured by the Brief Cope, than participants assigned to the neutral writing group.

Aim 5. Identify the relationship between written EE and active coping, use of emotional support, instrumental support, positive reframing, planning, humor, and acceptance coping strategies in individuals who have sustained an mTBI.

Pearson correlation revealed a statistically significant relationship between depression scores and planning, $r(12) = -.653$, $p = .029$**.** It was found that as depression scores decreased, planning scores increased. T-tests were accomplished to determine the differences between the N and EE groups when investigating active coping strategies, use of emotional support, instrumental support, positive reframing, planning, humor, and acceptance coping scales of the Brief Cope. Significant differences between the neutral and EE writing groups were found at the completion of the three writing trials in the planning and acceptance indices, $t(12) = 1.07$, $p = .05$ and $t(12) = .920$, $p = .019$, respectively. When examining the means of these two groups on these two coping indices, counterintuitively, it appeared that the N group obtained higher scores than the EE group

on both of these indices. The following table depicts the mean scores obtained for each index in each group.

Table 1: Planning and Acceptance Coping Scores Following Writing Trials.

	Planning	Acceptance
N Group	6.20 (1.3)	6.20 (.44)
EE Group	4.86 (2.5)	5.43 (1.8)

Post-Writing Follow-up

When the N group was compared to the EE group to examine participants' ratings about how personal their narratives were, the findings were not significant. Responses to open-ended questions on this post-writing questionnaire were reviewed. The following comments were made by the EE group. To questioning about whether the study was helpful, several responses included, "Yes, [it] has made me see how well I am doing"; "Yes, [it] reduced some stress"; and "Yes, I got to write about what I was feeling so I got it out and feel better afterwards not right after, but after." Some of the comments by the N group for the same question ranged from no comment at all to the following, "No, it was just questionnaires." and "Yes, I am now interested in finding a neurologist."

Benefits Resulting from the Injury: Primary/Secondary Gain

The mTBI population is often associated with litigation, which raises questions about exaggeration of symptoms and protraction of symptom course for secondary gain. Military members, who comprised the sample investigated in this study, can not lawfully bring suit against the federal government. However, the possibility of military members feigning illnesses or disorders to evade work duties or be removed from military service is present. As part of this study, participants were required to complete face-valid questionnaires that relied on self-report

and information obtained from medical records, which relied heavily upon self-report as well. Therefore, it was expected that the data obtained in this study was vulnerable to the exaggeration of symptoms. With the exception of utilizing questionnaires that have little face-validity, it is difficult to objectively detect malingering on self-report measures. Instead, during the initial phone interviews with participants, they were asked an open-ended question about ways they may have benefited from their injuries. This question was asked to gain more insight into whether participants believed they could or would be compensated for their injuries above and beyond their military benefits. Specifically, each participant was asked, "How have you benefited from your concussion?" Most participants denied having benefited at all from their concussion or injuries. Two participants stated they were confused by the question, further stating that they did not know how to answer the question except to respond that they had not received or noticed any benefits. Given the responses obtained, this question was not the most effective at gaining insight into ulterior motives associated with seeking medical assistance or exaggerating symptoms for compensation. Of the 13 completers, all of them were still undergoing the medical board process at the time of their participation in this study. One factor that may have mitigated participants' motivation to exaggerate was that they were all informed that data obtained through this study would not affect their medical benefits and would not be shared with their medical providers. Perhaps future studies will investigate more objective ways to assess malingering in this context.

Post-hoc Analyses

Low power prevented the ability to disconfirm the null hypothesis in the majority of the statistical analyses proposed and accomplished. Therefore, it may have appeared that the emotional expression writing exercises had no effect on the outcome measures when, in fact, there was an effect (Cohen, 1994). To better determine the impact, if any, the writing exercises may have had on participants, post-hoc exploratory analyses were undertaken. Perhaps if each participant was reviewed as a separate case, these case analyses might yield useful information that could help shape future studies on this subject. The experimental group (EE group) was

further analyzed. The following will outline the characteristics of participants who demonstrated any decrease in depression scores on the NFI (responders), as hypothesized and the characteristics of participants who demonstrated no change in their depression scores on the NFI (non-responders). Scores on the depression scale of the NFI ranged from mild to moderate. Score, high or low, was not a factor in recruiting or assigning participants to each group. Therefore, participants were classified as responders or non-responders based on the direction of change in the score—and not by the amount of change observed or by an objective decrease in severity.

Five of the seven participants in the EE group demonstrated decreases in their scores on the depression scale of the NFI. Score changes ranged from 1-9 points. Two participants in the EE group did not respond, demonstrating increases in their scores on the depression scale. These score changes ranged from 2-8 points. It is noteworthy that all of the participants in the N group demonstrated increases in their scores on the depression measure. However, this group demonstrated the smallest range of change in scores ranging from 1-4.

Qualitative Writing Analysis. The narratives of those who benefited from the writing trials (responders), as demonstrated by decreases in depression scores on the NFI, were qualitatively compared to the narratives of participants who did not appear to benefit from the writing exercise (non-responders). Through this analysis, three main descriptive factors for the narratives emerged, which appeared to differentiate the responders from the non-responders. The first of these factors is the length. Narratives appeared to differ in length overall, based on a simple word count. Non-responders' narratives (n=2) wrote more than 100 words more per writing trial than responders. Review of their narratives also showed differences between the two groups in lengths over the three trials. Specifically, the responders' narratives appeared to be shorter than the non-responders' overall. Also, little change in length over time was noted in the narratives of the responders, while the non-responders' narratives appeared to increase in length over time.

The second difference is the recall of memory for the event. It appeared that all of the responders either claimed little or no memory for the event that resulted in their head injuries or did not recount the trauma in detail at all. They understood on an intellectual level what caused their injuries through knowledge of what they were doing prior to their accidents and through others' recall of their accidents. However, responders would note in their narratives that they did not have a clear memory of the event. In contrast to this, both of the two non-responders appeared to spend much of their narratives recalling the events that caused their injuries in detail—much of which contributed to the length of their narratives.

Lastly, responders, as instructed, appeared to recount the positive aspects of their injuries, often including thoughts about their support networks. For instance, responders often mentioned the value of their families or depicted their families' role in the recovery process. Thoughts about support networks, including family, were, for a large part, excluded from the non-responders' narratives. Instead, there appeared to be more negative thoughts represented in these narratives than were observed in responders' narratives. Additionally, these narratives were self-focused, focused on the traumatic injuries, and appeared to exclude thoughts and feelings about factors that have helped them cope with their traumas, all of which was noted in the responders' narratives. The following table includes quotes from the narratives that reflect what was observed throughout participants' writings.

Table 2: Writing excerpts: Responders versus non-responders

Responders	**Non-responders**
- "I found out that getting back into a routine really helped me out. I also set goals for myself that took my mind off of my injuries and gave me something to focus on. All of my feelings of resentment left me, as well as feeling sorry for myself. Although my injuries ended my military career, I realized that this was the beginning of a new life and that 20 years in the military was not meant to be."	- "I hear people complaining about little things and it makes me so mad. They complain how they have to work so much. What about the soldiers in Iraq who work 18-20 hours a day? These stupid people have nothing to complain about." - "I am not who I used to be- not physically or emotionally. I just want to be me again. I cry every damn time

- "Feelings are feelings, it's just part of living. What happens to me and how I feel it's what I make it." - "I can look at old pictures of me and still feel happy about things. I miss my arm and it would definitely make things easier, but what can I do? I think things are really positive right now in my life and I hope they stay that way." - "I try to remain strong in front of those around me. I use their worries and concerns as a catalyst to strengthen myself inside." - "I have a hard time remembering things and doing things each day is a struggle…, but I know that I got my friends and family down here which that is ok."	I think about all the stuff and all of the guys that were hurt with me. They don't seem to be taking it all of this very hard…But I still have emotional pain and physical pain and there seems to be no way to fix it."

Chapter 10: Discussion

In summary, it was found that at baseline, participants who chose to complete at least the first three trials of this study had a higher PTA time and were more likely to have been injured through an IED blast. Although few significant findings emerged due to low power, data trends were noted for outcome and process measures. Specifically, when the EE group was compared to the N group, the EE group evinced an average one-point drop in depression in comparison to no changes among participants in the N group, whose depression scores increased or remained the same. This aspect of the data is obviously not significant. Therefore, little confidence can be ascribed to it. However, if viewed as part of a pattern or trend, it loosely falls in line with Hypothesis 1. Trend data also suggested that the writing exercises appeared to have some effect on participants. The N and EE groups appeared to respond antithetically in how they scored on measures of positive and negative affect following each trial. At baseline, two correlations were found, as predicted. Depression scores were positively correlated with PCS scores and behavioral disengagement coping style. These differences disappeared following the completion of the three writing trials, which may be meaningful or, again, may be reflective of statistical vulnerability when the sample size is low. Following the writing trials, negative correlations were found between depression scores and planning. However, against expectations, the neutral group appeared to have higher planning and acceptance coping scores than the EE group.

Although not completely clear, it is possible that those who completed this study may have perceived themselves as having had severe injuries that required intervention. Possible findings suggest the 13 participants who completed the majority of the study (completers) may have experienced or perceived more significant consequences from their injuries than those who did not complete or return study materials (non-completers). This interpretation is derived from data suggesting that average PTA time and average report of LOCs were higher among completers than among non-completers. Further, it appeared that the source of injuries varied in non-completers, while 92% of completers' injuries were obtained through IED blasts.

Completers' decisions to complete study procedures may have resulted from self-selection for the study due to perceived injury severity. That is, completers may have believed themselves to be more severely injured than the average participant and, as a result, they needed additional services or focused attention on their concerns. Another possible explanation for completers appearing more severely distressed is that these participants may have actually been more severely injured than non-completers, and, as a result, these participants may have had more time available to complete the study. Perhaps IED blasts produce greater neurobehavioral consequences at the time of the injury, which leads to actual increased injury severity. That is, completers may not have returned to work or may not have been working as rigorously as non-completers due to injury severity and, as a result, these participants had time to follow-through with the study's procedures.

As noted, the necessary power was not obtained to definitively test the hypotheses outlined. Some significant findings were revealed, including relationships between depression scores and PCS scores and coping indices at baseline and following the three writing trials. However, in most cases statistical analyses yielded little information. Trend data, outlining the direction that scores tended to follow, suggested that the EE writing exercises may have impacted immediate mood, as well as depression symptoms. The evidence that EE groups consistently showed a "different" pattern of change when compared to the N group suggests that the nature of the writings had some impact on participants. Although not yielding statistically viable data, EE participants' qualitative responses on feedback forms suggested this as well. Based on open-ended responses about how helpful this study was, the EE group mostly indicated that the study was helpful at improving stress or mood, while the N group commented that the study either was not helpful or was helpful at helping them plan or organize, or they did not comment at all.

Whether EE exercises result in long term changes in depression symptoms in a head-injured population needs to be further investigated with a larger sample. Nevertheless, two significant results emerged when the relationships between coping and depression scores were

analyzed. As would be expected based on the literature and what was proposed, baseline depression scores positively correlated with behavioral disengagement. That is, the higher the depression score the more likely one was to be behaviorally disengaged. Depressed mood may impact one's motivation to engage in typical, pleasurable, or activities that lead to a sense of accomplishment. Behavioral activation is often prescribed by cognitive-behavioral therapists to counteract the downward spiraling of mood when one becomes disengaged (Gurman & Messer, 2003). This finding disappeared post-writing, which is likely due to the reduction in power, as power is vulnerable in repeated measures analyses. The initial findings at baseline confirmed the behavioral consequences of depression.

Additional relationships in coping and depression emerged; planning and acceptance indices post-writing were significantly different between the two groups. These findings suggest that the score differences surpassed the challenges in power that were observed throughout the analyses. Counter to initial expectations, the N group obtained higher scores in planning and acceptance. Reasons for this are unclear. However, it may be that the neutral writing assignment that required participants to write about the details of their days was responsible for the more superior planning scores than for the EE group. Perhaps one may consider that mTBI patients may improve organizational/planning skills by engaging in concrete exercises that focus their attention on planning while excluding confounding factors like feelings or emotion. The implications of this finding reach beyond the parameters of the current study. With regards to the higher acceptance scores, it is unclear what may have been responsible for this finding. Nevertheless, a negative correlation emerged when the relationship between depression and planning was investigated, which was in line with expectations. Regardless of the score, high or low, as depression scores increased planning scores decreased and vice versa.

Lastly, as expected, depression scores positively correlated with PCS symptoms. This information confirms previous research that documents the strong relationship between persisting depression and persisting PCS. Which comes first or which leads to the other is not well

understood. However, it is acceptable to surmise that reducing depression may also positively impact one's report or experience of PCS symptoms, which was the focus of the current study.

Responders versus Non-responders

One purpose of this study was to clarify the clinical utility of the emotional expression paradigm. The population investigated in the current study was clinical in at least two ways: they were depressed and head-injured. The literature supports EE's effectiveness in reducing depression and improving health outcomes, in various clinical populations from rheumatoid arthritic to cardiac populations. No study to date, however, has attempted to determine the effectiveness of EE in a dual clinical population. Although the current study made an attempt, inadequate sample size precluded this question from being answered fully. Despite challenges to interpret statistical data, post-hoc analyses yielded three factors that appeared to differentiate between the narratives of responders and non-responders: 1) narrative length, 2) self-reported recall of memory for the event that caused the injury, and 3) perceived social support/positive outlook. However, interpretations of these findings are speculative at best and have been approached with caution. If viewed as case analyses, patterns and trends noted in the outcome measures and the narratives may suggest that the EE exercise may have had an effect on participants.

Perceived Benefits

As both groups wrote about their thoughts and feelings, perhaps emoting simply is not enough in this specialized population. Qualitative review of the participants' narratives revealed that those who benefited closely followed the self-regulatory prompt, as instructed. It is unclear exactly the reasons the differences in writing quality emerged. Perhaps the non-responders demonstrated more cognitive rigidity due persisting deficits associated with their head injuries and they required more specific cueing. Nevertheless, there are three theories to consider why the self-regulation prompt may have resulted in the differences between the responders and the non-responders. The first is that responders actually benefited from the writing because they spent

more time exploring the positive aspects of their trauma experiences. That is, they explored the "perceived benefits" of their head injuries (King & Miner, 2000). This may have resulted in emotional and cognitive self-regulation that produced depressed thinking and emotion. The other is that, as asserted in cognitive-behavioral theory, non-responders maintained depressed mood because they rehashed negative cognitions and did not gravitate from their negative thoughts and emotions to the self-regulatory prompt, as instructed. A last consideration is that non-responders' memories for the event were so prominent that they may have required more time to gravitate to the self-regulating phase of their writings. Perhaps more writing time would have guaranteed that all participants in the EE group would have explored a balanced view of their traumas (negative emotion and perceived benefits), allowing even the non-responders to positively reframe thoughts, which cognitive-behavioral theorists and therapists believe will reduce depression (Beck, 1995; Ellis & Grieger, 1986; Gurman & Messer, 2003; Walen, DiGiuseppi, & Dryden, 1992).

As initially hypothesized, the self-regulation prompt was included in the experimental writing instructions (writing trials 1-3) to increase the effectiveness of the writing exercises in the current population. Responders appeared to adhere to and hone in on the writing instruction's prompt to "…include in your writing some things that you can do that may help you deal with the problems you have had..." Moreover, responders' narratives appeared to include fewer negative comments about their injuries and did not include many of details about their injuries, which are often believed to contribute to the surge in negative affect observed, when compared to the narratives of non-responders. Lastly, responders' narratives included details about family and social support systems that have emerged since their injuries—something that was deficient in the essays of the non-responders. Unlike in single clinical, sub-clinical, or non-clinical populations where the original Pennebaker-derived instructions have been effective at improving various outcomes, including mood, it may be that in dual-clinical populations this prompt is necessary to reduce depression. More specifically, the prompt may be helpful at reducing depression in a head-

injured population. Perhaps, recognizing and focusing on perceived benefits of a trauma bolsters patients' positive self-appraisal and self-efficacy especially when there is an absence of face-to-face interaction in a complex population. Therefore, self-regulation prompts to focus on perceived benefits may need to be the primary focus of future writing instructions. This is similar to the findings in King and Miner's (2000) study that investigated the effects of writing about perceived benefits on health. King and Miner explored the effectiveness of writing about perceived benefits compared to the typical Pennebaker (1985) emotional expression and neutral writing groups. Those who explored perceived benefits in their writings experienced very similar results to the typical emotional expression writing group and both techniques were more effective at reducing health center visits than the neutral group task. Similar to the results of that study, it is possible that the self-regulatory prompt led participants to explore the meaning associated with their traumas more than the just the trauma itself. The rationale that was proposed is that promoting self-regulation allows participants to structure and explore their trauma without focusing solely on the obvious distress. This allows participants to explore positive factors that have helped them through their trauma and may actually guide their ability to cope at the time of recall. Further, one has the opportunity to develop ideas about positive outcomes or perceived benefits that evolved as a result of the trauma, which may assist participants in developing effective cognitive reframes and flexible cognitive appraisals that improve factors related to good mood (hope, optimism, etc.). Much of this is often accomplished in face-to-face therapy and is associated with good outcomes (i.e., reduction in psychological consequences) (Gurman & Messer, 2003). Altogether, focusing on perceived benefits may help one to develop and foster meaning from a traumatizing situation. King and Miner (2000), Pennebaker and King (1999), Amir, Stafford, Freshman, and Foa (1998), among others, assert that deriving meaning from an otherwise fragmented trauma memory may be a mechanism of change within the emotional expression paradigm.

While developing the methods for this study, some questions were raised about the type of writing exercise that would be helpful to reduce depression in a head-injured population. The

typical emotional expression paradigm with the inclusion of Cameron and Nichols (1998) self-regulation prompt was decided upon based on the effectiveness of these procedures in multiple populations, healthy, sub-clinical, and clinical alike. The results of the current study may not have clearly outlined the effect of EE on depression following a head injury. However, these findings may help inform the structure of future writing procedures and instructions in this population or any dual or complex clinical population.

Cognitive Theory of Depression

It is hypothesized that the self-regulatory instruction prompted participants to engage in thoughts and feelings about perceived benefits, which may have resulted in the decreased depression scores. The two non-responders did not appear to explore this aspect of the writing instructions. Instead, their writings, as also instructed, focused on the thoughts and feelings regarding their traumas. In previous studies, exploring traumatic events, experiencing surges in negative emotion words in the writing, and demonstrating clear memories for traumas usually resulted in positive outcomes. However, the opposite of this was found in the current population. Not only was lack of benefit noted, non-responders' depression scores actually increased. Beck (1995), Ellis and Grieger (1986), and Walen, DiGiuseppi, & Dryden (1992), among other cognitive theorists assert that emotions are maintained by one's cognitions and/or beliefs or, in other words, one's interpretation of an event. Specifically, depression is maintained by distorted, negative thinking. This was previously described by Beck (1976) through one illustration, the negative triad. The negative triad describes thinking negatively of oneself, the world, and the future, which possible causes depression to persist. Perhaps disclosing trauma memories in a head-injured population does not extinguish negative interpretations and negative cognitions associated with the trauma. Instead, it preserves them. As a result, depressed mood persists. More specifically, it may be that not adhering to the self-regulation prompt that may encourage one to move from construing the trauma as negative may maintain or worsen depressed mood. It is unclear why the two non-responders fixated on this aspect of the writing exercise. It should be

considered that the intact memory of the trauma resulted in greater recall, which prevented participants from gravitating to the self-regulation prompt in their writing. If this is the case, it may be more useful to have future patients engage in writing about the perceived benefits of their trauma and not consider the negative thoughts and feelings related to the trauma at all. Although statistically unsupported, it appears that this may be a contributing factor to how responders appeared to derive benefit. It is possible that disclosing real trauma memories in a time-limited writing exercise without specific directions or structure may not be helpful for all depressed groups. Lastly, non-responders were few. It is possible that if an adequate sample size had been investigated, it would have rendered these findings as statistically insignificant findings or outliers.

Inadequate Processing Time Theory

A last theory to consider is that the non-responders simply did not have the time to engage in self-regulation of their emotions, due to their significant recall of their traumas. Perhaps if the procedures included additional trials, a benefit would have been observed. It appeared non-responders were "stuck" in the negative affect produced by their trauma memories. King and Miner (2000) documented a similar finding when they compared a combined procedure with trauma-only and perceived benefits-only writing groups. The combined groups failed to reach the same positive outcomes that were observed in the trauma-only and perceived benefits-only groups. Participants must prioritize what the focus of their narratives will be in a time-limited, trial-limited exercise. There is a possibility that instructing participants to attend to both trauma iteration and self-regulation precluded some members, particularly the non-responders, from processing their thoughts and emotions fully enough to benefit. Greenberg, Wortman, and Stone (1996) found that when they asked college students to complete a one-time writing exercise using either a real trauma or imaginary trauma, both groups demonstrated improved health. However, the real-trauma group reported more fatigue and increased avoidance. This group also had higher scores on depression measures when compared to the imaginary trauma group. The most likely

explanation for this finding is that this group disclosed real traumas, compared to the group that wrote about fictional events. Nevertheless, in the typical emotional disclosure exercise, one typically discloses about a real trauma over three to four occasions, which results in positive outcomes. Given Wortman and Stone's deviation from the typical disclosure paradigm, it is possible that in this study the time limit placed on the participants' ability to disclose about real traumas was related, in some way, to the higher levels of fatigue, avoidance, and depression reported by the participants in this group. As the literature base regarding the factors that render EE effective grows, it may become more clear which clinical populations require more or less time to obtain benefit from the writing exercises. Further, more information about how long each individual writing exercise should last to produce an effect will become clearer. Again, as researchers attempt to clarify the mechanisms responsible for the effect writing has on depression or any outcome index, it is useful to know that instructions may need to be unimodal and simple enough to explore in a time- and trial-limited writing protocol, especially in a head-injured, depressed population.

Limitations of the current study

One obvious limitation to the current study is the attrition due to participants' not returning study materials through the mail. This may have been a combination of an expected phenomenon, as it is expected that a percentage of participants may forget to mail study materials or may lose them prior to returning them, and several challenges that may arise in telephonically managed procedures. The AI exerted significant effort obtaining this information, using all possible options (i.e., phone, mail, and email). Nevertheless, as participation in this study was an added responsibility to already existing responsibilities (e.g. work, family, actual medical appointments), this may have been an artifact of the population chosen to participate. Additional challenges that were unique to this population were deployments and relocations that, at times, were reported to occur on short notice. Because of these challenges, materials had not been mailed prior to departure or, in some cases, materials were believed or reported to have been

mailed without participants' certainty (i.e., lost). Although the procedures of this study may be replicated anywhere, this study's assumptions and findings can only be generalized to a military population, which is another limitation to this study. The initial goal was to attract any DoD beneficiary, which includes civilians and non-military family members. However, civilians did not make up a significant portion of this sample because the current patient population at the DVBIC is swelled with military members due to battle injuries. Given that recent referrals to the DVBIC have been well-represented by military members, it impacted the gender pool. Military personnel (specifically Army and Marine) are mostly male. Also, female soldiers are excluded from front-line activity, which is where most of the blast injuries likely occurred. Therefore, this study may only be generalized to the male military population.

Many of this study's limitations revolved around the unique battle climate that has emerged over the last four years. At the time this study was conceptualized and the population was chosen, it was not known that the current war would result in the numerous referrals to the DVBIC for mTBI due to blast incidents (e.g. improvised explosive devices, mortars, etc.). The pool that was used to recruit patients was representative of this. Moreover, the heightened operations during this climate have resulted in frequent deployments of military service members to combat regions and atypical medical processing (changes in timelines for medical boards/medical discharges and separations) for returned service members. These factors placed a high demand on service members who were appropriate for this study and, as a result, may have impacted their ability to be available to complete study procedures. Lastly, military members are at increased risk to develop post-trauma responses, including anxiety disorders like post-traumatic stress disorder (PTSD) due to the exposure to traumatic and/or life threatening experiences during war and hostile conflicts. It is estimated that one to four percent of service members exposed to combat endorsed experiencing symptoms associated with PTSD. These estimates are variable due to the amount of exposure to combat, the assessment instrument/modality, among other variables (Madigan Army Medical Center; United States Air Force). The incidents that preceded members'

TBIs were likely traumatic and likely increased these service members' risk to develop PTSD or symptoms associated with it. Service members are surviving combat incidents due to expedient medical care. As such, long-term effects of multiple injuries and emotional consequences, like PTSD, are becoming prevalent (Gaylord, Cooper, Mercado, Kennedy, Yoder, Holcomb, 2008). Given this increased risk, the likelihood that symptoms associated with not only depression, but also anxiety or PTSD is high in the sample used for this study. This study did not look at the impact that symptoms associated with PTSD may have had on members. The interaction between mTBI and PTSD was also not looked at. However, given the importance of these two issues in the population, it may need to be explored further in future studies.

Telemental health and other telehealth protocols are becoming increasingly popular as cost-effective ways to manage clinical populations (Barry, 2000; Bleich, 1998; Martin, Coyle, Warden, & Salazar, 2003; Morrison, Bergauer, Jacques, Coleman, & Stanziano, 2001). Telehealth models provide convenient support and treatments for patients in their home while freeing actual provider hours. These models have been used successfully to treat and monitor various clinical populations and are now even considered useful in mental health populations. Specifically noted earlier in this study is the effectiveness of telehealth in head-injured patients. Nevertheless, this study encountered several challenges while using this model, which resulted in the low number of participants who not only completed, but also returned, study materials. For instance, phone contacts were time-intense, due to participants missing initial calls or needing to reschedule. Future telehealth models may consider setting up research participation as an actual medical appointment, much like what would be accomplished in a research center, and not as a convenience. This model was chosen to investigate a cost-effective, time-efficient model that would reduce patients' contacts with medical brick-and-mortar institutions. The potential for the model to be effective in these manners is high. However, this study was not effective at capturing this, because the telephone appointments reflected an additional burden on the participants. To

better test this model, it may be useful to study patients who had few medical obligations or who are artificially available for study (i.e., on a medical hold unit).

For the purposes of the current study, some challenges may have been overcome by face-to-face visits with the AI at the start and end of the study. At least during the investigative phase, brief appointments would have been useful at capturing study materials with less time spent on both the AI's and participants' parts. Alternatively, a full-time, dedicated staff member would have been useful to devote appropriate number of work hours/telephone monitoring to accommodate any and all participants' schedules. It is believed that this model could be effective at providing a practical treatment to reduce depression in a fiscally efficient, time-effective manner. However, to further investigate the effectiveness and practicality of this model, it may be best to do this without a significant restraint on the time to accomplish the study in a setting with dedicated resources for clinical research.

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Appendix A: Writing Instructions for the EE and N groups for Each Trial

(Experimental Group) Writing Instructions for Day 1:

For the next 20 minutes, I would like for you to write about your deepest thoughts and feelings about your concussion. You may consider writing about your feelings concerning the accident or event that led up to your injury; the effect that the injury has had on your life currently; difficulties you have encountered following your concussion; how the injury has impacted your family or relationships; or any topic that includes how you feel about your concussion. Also, you may write about your feelings about the past, present, or future. Please include in your writing some things that you can do that may help you deal with the problems you have had. In your writing, I'd like for you to really let go and explore your very deepest emotions and thoughts. The only rule for your writing is that you write continuously for 20 minutes. Do not worry about grammar, spelling, or sentence structure. Just write. If you run out of things to write about, this is ok. Just continue to write, re-writing thoughts that you have already written about until the time is up. Remember, all of your writing will be completely confidential.

(Experimental Group) Writing Instructions for Day 2 and Day 3:

Again, I would like for you to write for the next 20 minutes. Similar to your first writing, I would like for you to write about your deepest thoughts and feelings about your traumatic concussion. Like before, you may consider writing about your feelings concerning the accident or event that led up to your injury; the effect that the injury has had on your life currently; difficulties you have encountered following your concussion; how the injury has impacted family or relationships; or any topic that includes how you feel about your head injury. Please include in your writing some things that you can do that may help you deal with the problems you have had. Have you tried the things that you thought could be helpful to deal with your problems? If so, were they helpful? Also, you may write about your feelings about the past, present, or future. In your writing, I'd like

for you to really let go and explore your very deepest emotions and thoughts. Remember, the only rule for your writing is that you write continuously for 20 minutes. Do not worry about grammar, spelling, or sentence structure. Just write. Once again, if you run out of things to write about, this is ok. Just continue to write, re-writing thoughts that you have already written about until the time is up. Remember, all of your writing will be completely confidential

.**(Neutral Group) Writing Instructions for Day 1:**

For the next 20 minutes, I would like for you to write about what you have done since you woke up this morning. Please describe in detail any event that has happened today. Include in your writing what happened, at what time, how long it lasted, etc. The important thing is that you describe every aspect of the event. Please do not include any thoughts or feelings. Write objectively about what you have done today. Please write continuously for 20 minutes. Do not worry about grammar, spelling, or sentence structure. Just write. If you run out of things to write about, this is ok. Just continue to write, re-writing what you have already written about until the time is up. Remember, all of your writing will be completely confidential.

(Neutral Group) Writing Instructions for Day 2 and Day 3:

Again, I would like for you to write for the next 20 minutes. Similar to your first writing, I would like for you to write about what you have done since you woke up this morning. Please describe in detail any event that has happened today. Include in your writing what happened, at what time, how long it lasted, etc. The important thing is that you describe every aspect of the event. Please do not include any thoughts or feelings. Write objectively about what you have done today. Please write continuously for 20 minutes. Do not worry about grammar, spelling, or sentence structure. Just write. If you run out of things to write about, this is ok. Just continue to write, re-writing what you have already written about until the time is up. Remember, all of your writing will be completely confidential.

Appendix B: Outline of Study Procedures

Recruitment	Trial 1 (Week 1) Day 1	Trial 2 Day 2	Trial 3 Day 3	Week 6
➢ Recruit Participants via phone- ➢ Administer Screening measures: -DSM Checklist -Descriptive Info. (15 minutes)	➢ Informed Consent ➢ Baseline/Outcome Questionnaires -Post mTBI Checklist - The Brief Cope - NFI ➢ Randomly assign participants to groups ➢ PANAS ➢ First Writing Trial ➢ PANAS ➢ Writing Manipulation Questionnaire ➢ Provide instructions for remainder of materials (1.5 hour)	➢ Phone reminder ➢ PANAS ➢ Second Writing Trial ➢ PANAS ➢ Writing Manipulation Questionnaire (25 minutes)	➢ Phone reminder ➢ PANAS ➢ Third Writing Trial ➢ PANAS ➢ Writing Manipulation Questionnaire ➢ Baseline/Outcome Questionnaires -DSM Checklist - Post mTBI Checklist - The Brief Cope - NFI (45 minutes)	➢ Phone reminder ➢ Follow-up questionnaires administered by phone/mail -DSM Checklist -Post mTBI Checklist -The Brief Cope -NFI -Writing Manipulation Questionnaire (20 minutes)

Appendix C: Brief Cope

The Brief Cope

These items deal with ways you've been coping with the stress in your life since you sustained your head injury. There are many ways to try to deal with problems. These items ask what you've been doing to cope with this one. Obviously, different people deal with things in different ways, but I'm interested in how you've tried to deal with it. Each item says something about a particular way of coping. I want to know to what extent you've been doing what the item says. How much or how frequently.

Don't answer on the basis of whether it seems to be working or not—just whether or not you're doing it. Use these response choices. Try to **rate each item** separately in your mind from the others. Make your answers as true FOR YOU as you can.

1 = I haven't been doing this at all
2 = I've been doing this a little bit
3 = I've been doing this a medium amount
4 = I've been doing this a lot

1. I've been turning to work or other activities to take my mind off things. ____
2. I've been concentrating my efforts on doing something about the situation I'm in. ____
3. I've been saying to myself "this isn't real." ____
4. I've been using alcohol or other drugs to make myself feel better. ____
5. I've been getting emotional support from others. ____
6. I've been giving up trying to deal with it. ____
7. I've been taking action to try to make the situation better. ____
8. I've been refusing to believe that it has happened. ____
9. I've been saying things to let my unpleasant feelings escape. ____
10. I've been getting help and advice from other people. ____
11. I've been using alcohol or other drugs to help me get through it. ____
12. I've been trying to see it in a different light, to make it seem more positive. ____
13. I've been criticizing myself. ____
14. I've been trying to come up with a strategy about what to do. ____
15. I've been getting comfort and understanding from someone. ____
16. I've been giving up the attempt to cope. ____
17. I've been looking for something good in what is happening. ____
18. I've been making jokes about it. ____
19. I've been doing something to think about it less, such as going to movies, watching TV, reading, daydreaming, sleeping, or shopping. ____
20. I've been accepting the reality of the fact that it has happened. ____
21. I've been expressing my negative feelings. ____
22. I've been trying to find comfort in my religion or spiritual beliefs. ____
23. I've been trying to get advice or help from other people about what to do. ____
24. I've been learning to live with it. ____
25. I've been thinking hard about what steps to take. ____
26. I've been blaming myself for things that happened. ____
27. I've been praying or meditating. ____
28. I've been making fun of the situation. ____

Appendix D: DSM-IV-TR Checklist for Major Depressive Disorder

DSM-IV-TR Checklist for Major Depressive Disorder

A. Please check off the following symptoms if present in a 2-week period prior to interview and represent a change from previous functioning.

Yes □	No □	**Depression Symptoms**
□	□	***Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful)***
□	□	***Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others)***
□	□	Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day.
□	□	Insomnia or hypersomnia
□	□	Psychomotor agitation or retardation
□	□	Fatigue or loss of energy
□	□	Feelings of worthlessness or excessive or inappropriate guilt
□	□	Diminished ability to think or concentrate, or indecisiveness, nearly every day
□	□	Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

These symptoms cause distress in social, occupational, or other important areas of functioning.
Yes □ **No** □

Appendix E: Descriptive Information Questionnaire

Descriptive Information Questionnaire

Personal Background:

1. Date of Birth: _____

1a. Age_____

2. Gender: □Male □Female

3. Ethnicity (Check all that applies)

□ White, not Hispanic origin □ Hispanic
□ Black, not Hispanic in origin □ American Indian
□ Asian or Pacific Islander □ Other_____

3a. Is English your first language? □YES □ NO
3b. Do you write/speak English fluently? □YES □ NO

Education:

4. What is the highest level of education you completed? (Check one)

□11th grade or below □High School/GED □2 years of College of Trade School □College Graduate or Professional Training

Concussion:

5. When did your most recent concussion occur? (Month/Year) _____

5a. How? (Check one)
□ Motor Vehicle Accident □ Fall □ Hit with Object
□ Assault □Combat □ Other:/Please list:____

5b. Experience loss of consciousness (LOC)? □YES □ NO If yes, for how long?_____
5c. Experience memory loss? □YES □ NO If so, for how long? _____
5d. Do **you** remember what occurred before the accident ? □YES □ NO
5e. Do **you** remember what happened immediately after? □YES □ NO
5f. Was your skull fractured? □YES □ NO
5g. How many days were you in hospital?: _____
5h. Did you sustain other physical injuries: If so, describe below:

_____

5i. How have you benefited from your concussion?:_____
5j. How many head injuries have you had in the past? _____
5k. Please describe what happened during previous head injuries:

_____

5l. When did these happen?:_____

Physical Health:

6. Do you have a chronic medical condition? □YES □ NO

If yes, please list: _____

7. Are you taking any medication? □YES □NO

If yes, please list: _____

Mental Health:

8. Please place a check next to any of the following that you have been diagnosed by a psychologist or psychiatrist?

- □ Bipolar disorder/ Manic Depressive
- □ Schizophrenia
- □ Substance Abuse (alcohol, illicit, or prescription)
- □ Learning Disability (e.g., dyslexia)
- □ Other: _____

9. Are you currently receiving any kind of counseling, psychological, or psychiatric services (e.g., therapy, group therapy)?□YES □NO

10. Are you currently taking medication for any psychological condition? □Yes □No

If yes, list medication:_____

10a. How long have you been taking this medication?: _____

10b. Expect any changes in medication?: _____ When?:_____

Compensation:

11. Are you undergoing a Medical Board?: □YES □ NO

12. Will you receive any compensation from your injuries?: □YES □ NO

If yes, list: _____

Appendix F*:* PANAS

PIN#_____

Trial:_____

PANAS

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you feel this way at this moment. Use the following scale to record your answers.

1	2	3	4	5
very slightly or not at all	a little	moderately	quite a bit	extremely

____ interested
____ distressed
____ excited
____ upset
____ strong
____ guilty
____ scared
____ hostile
____ enthusiastic
____ proud
____ irritable
____ alert
____ ashamed
____ inspired
____ nervous
____ determined
____ attentive
____ jittery
____ active
____ afraid

Appendix G: Post-writing Questionnaire- Process

Post-writing Questionnaire- Process

1) How personal or emotional was your writing?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

2) Did the writing cause you to feel strong emotions?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

3) Have you often thought about the problems that you included in your writing?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

Appendix H: Post-writing Questionnaire- Follow-up

Post-writing Questionnaire- Follow-up

1) How personal or emotional was your writing?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

2) Did the writing cause you to feel strong emotions?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

3) Have you often thought about the problems that you included in your writing?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

4) How valuable or meaningful do you feel this experience has been for you?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

5) Do you feel you have more control over problems related to your concussion?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

6) Did this study help you to deal with the challenges or problems that you face?

Not at all	A Little	Neutral	Somewhat	A Great Deal
1	2	3	4	5

7) Was this study helpful: Yes No If yes, how? _____.

If not, why do you think it was not: _____

Appendix I: Medical Record Checklist

Medical Record Checklist

Inclusion Criteria	☐ Hx of mTBI: How many? _____
	Loss of Consciousness ☐ **Y** ☐ **N** *Time:*_____
	Retrograde / Post-traumatic Amnesia ☐ **Y** ☐ **N**
	Change in Mental Status ☐ **Y** ☐ **N**
	Neurological deficit in absence of LOC greater than 30 minutes ☐**Y** ☐ **N**
	Glascow Coma Scale < 13 after 30 minutes ☐ **Y** ☐ **N**
	☐ 6 ≤ Post-injury
	☐ Depressed Mood or Anhedonia
Exclusion Criteria	☐ Sustained neurological hemorrhage
	☐ Skull Fractures
	☐ Hx of Severe Head Injuries
	☐ Current SI
	Frequency: ☐N/A ☐Never ☐ Rarely ☐Sometimes ☐Frequently
	Intensity: ☐Fleeting ☐Focused ☐Intense ☐Rumination
	Duration: ☐Seconds ☐Minutes ☐Hours
	Previous Attempts: ☐ **Y** ☐ **N** **How many?** _____
	☐ Medication Changes *When*: _____
	☐ Severe Psychiatric Disorder
	☐ Bipolar disorder/ Manic Depressive .
	☐ Schizophrenia .
	☐ Previous Dx of Alcohol Dependence .
	☐ Currently using illicit substances .
	☐ Developmental Disability .
	☐ Other: _____ .
	☐ Not fluent in English

	☐ Time inpatient:_____
	Any orthopedic injuries: ☐ **Y** ☐ **N**
	☐ Psychotherapy for depression or anxiety? : ☐ **Y** ☐ **N**

Appendix J: Post mTBI Symptom Checklist

POST MTBI SYMPTOM CHECKLIST
Center for Head Injuries, Johnson Rehabilitation Institute

NAME _____ DATE _____

AGE _____ SEX _____ EDUCATION _____

Please rate the following symptoms with regard to how much they have disturbed you *in the last two(2) weeks.*

0 = None - Rarely if ever present; not a problem at all.

1 = Mild - Occasionally present, but it does not disrupt activities; I can usually continue what I'm doing; doesn't really concern me.

2 = Moderate - Often present, occasionally disrupts my activities; I can usually continue what I'm doing with some effort; I feel somewhat concerned.

3 = Severe - Frequently present and disrupts activities; I can only do things that are fairly simple or take little effort; I feel like I need help.

4 = Very Severe - Almost always present and I have been unable to perform at work, school or home due to this problem; I probably cannot function without help.

1. Feeling dizzy:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

2. Loss of balance:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

3. Poor coordination, clumsy:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

4. Headaches:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

5. Nausea:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

6. Vision problems, blurring, trouble seeing:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

7. Sensitivity to light:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

8. Hearing difficulty:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

9. Sensitivity to noise:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

10. Numbness or tingling on parts of my body:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

11. Change in taste and/or smell:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

12. Increased or decreased appetite:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

13. Poor concentration, can't pay attention, easily distracted:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

14. Forgetfulness, can't remember things:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

15. Difficulty making decisions:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

16. Slowed thinking, difficulty getting organized, can't finish things:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

17. Fatigue, loss of energy, getting tired easily:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

18. Difficulty falling or staying asleep:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

19. Feeling anxious or tense:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

20. Feeling depressed or sad:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

21. Easily annoyed/irritability:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

22. Poor frustration tolerance, feeling easily overwhelmed by things:

0	1	2	3	4
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

23. Management of daily activities:

-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

24. Sexual functioning:

-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

25. Concerns or worries about health:

-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

26. Problems getting along with spouse:

-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

27. Problems getting along with family members:

-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

28. Participation in social activities:

-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

29. Participation in sports/recreation activities:

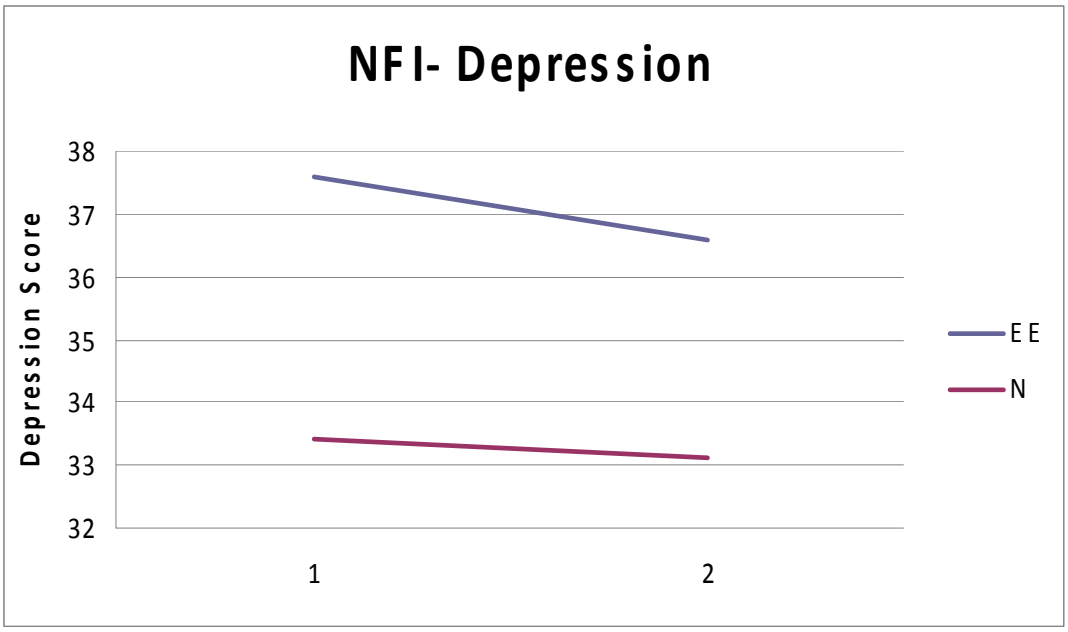
-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

30. Work and job performance:

-2	-1	0	1	2
NONE	MILD	MODERATE	SEVERE	VERY SEVERE

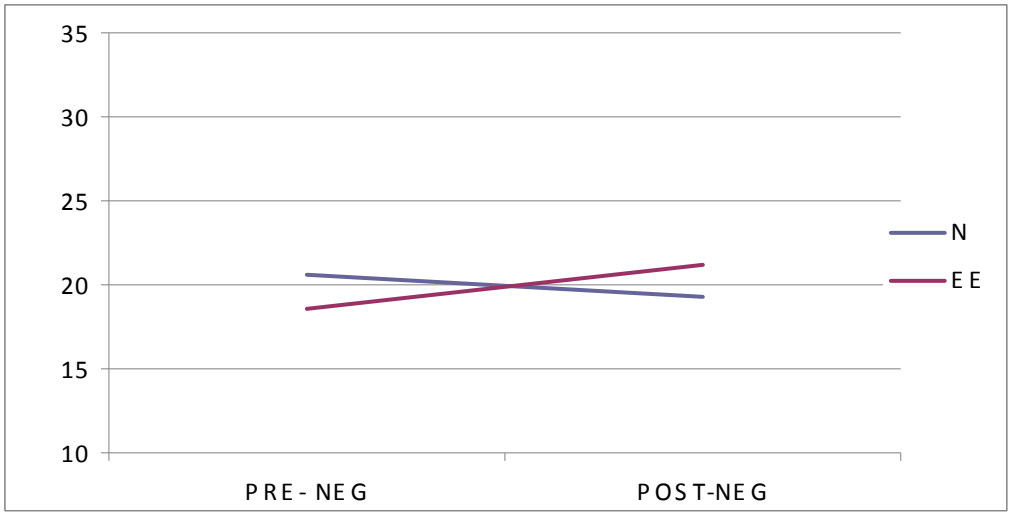
Appendix K: Graphs

Average Depression Scores

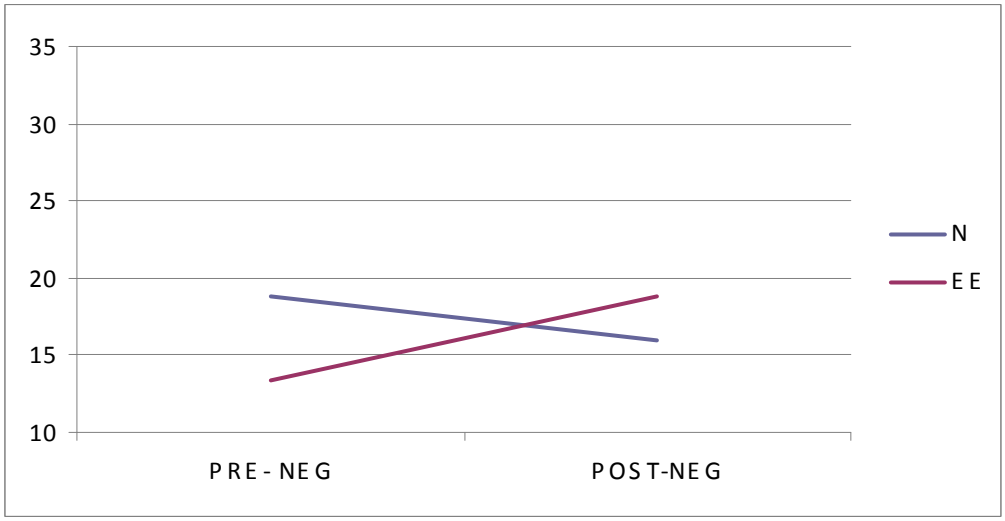


Graph 1: *Depression Scores Baseline to the Completion of All Writing Trials*

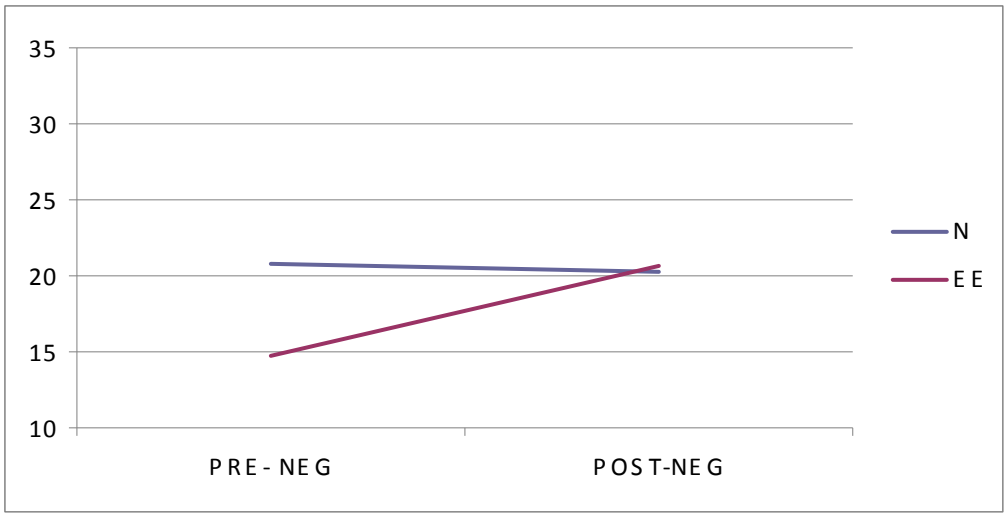
Average Pre- and Post-PANAS Scores for Negative Affect by Trial



Graph 2: *Negative Affect Pre- and Post-writing (Trial 1)*

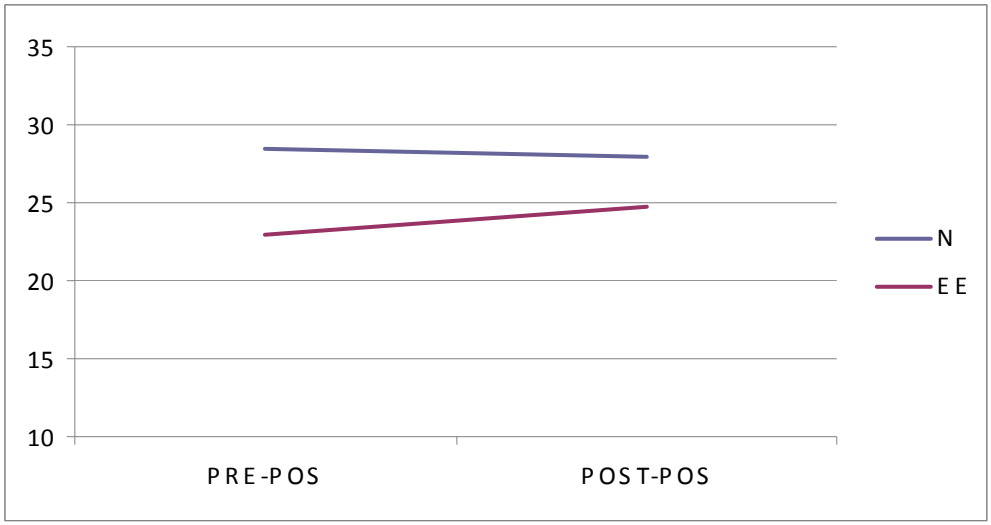


Graph 3: *Negative Affect Pre- and Post-writing (Trial 2)*

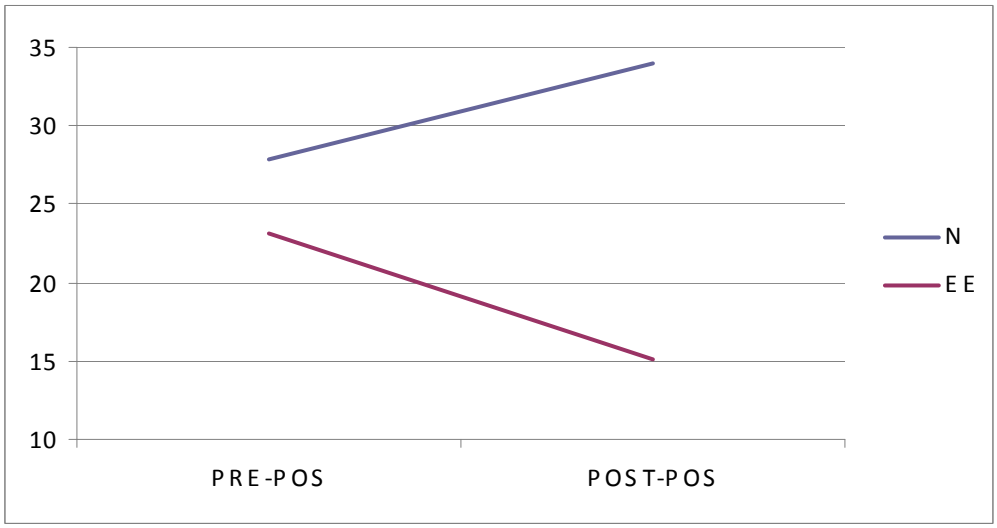


Graph 4: *Negative Affect Pre- and Post-writing (Trial 3)*

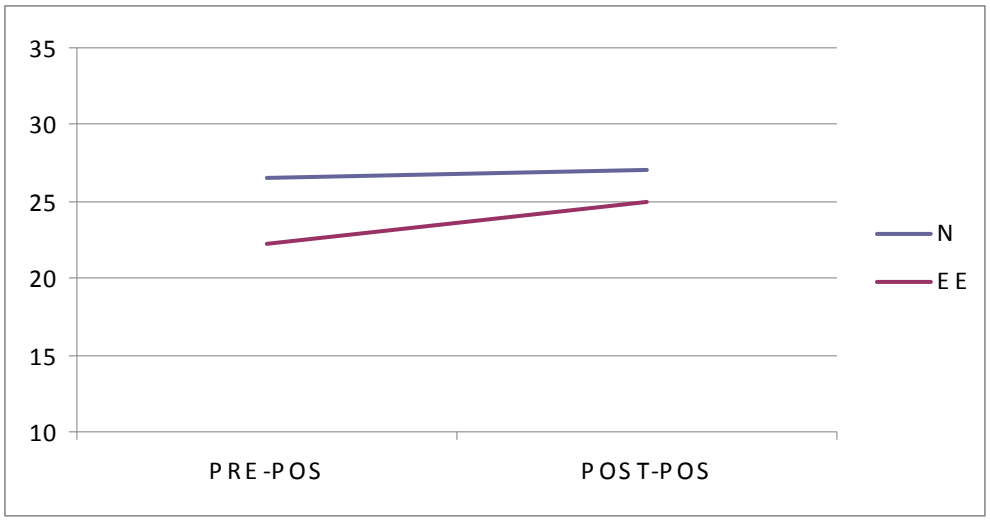
Average Pre- and Post-PANAS Scores for Positive Affect by Trial



Graph 5: *Positive Affect Pre- and Post-writing (Trial 1)*



Graph 6: *Positive Affect Pre- and Post-writing (Trial 2)*



Graph 7: *Positive Affect Pre- and Post-writing (Trial 3)*

Vita

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(Summary of experiences- Not inclusive)

Institutions Attended:

Drexel University	*(*2008) Doctorate of Philosophy	Clinical Psychology
	(2002) Master of Science	Clinical Neuropsychology
Temple University	(1998) Bachelor of Arts Cum Laude	Psychology/African American Studies

Publications:

Anderson, S. and Kloss, J. (2002). The relationship between depression and language use in stress-related narratives. Drexel University. College of Arts and Sciences. (Thesis)

Smith S., Kloss J., Kniele K., & Anderson, S. (2007). A comparison of writing exercises to motivate young women to practice breast self-examinations. *British Journal of Health Psychology, 12,* 111-123.

Teaching Experience:

Introduction to Psychology	Undergraduate (Teaching Assistant)
Sensory and Perception	Undergraduate
Teaching and Assessment	Undergraduate

Professional Experience:

2004-present *USAF Clinical Psychologist*
Positions Held: Mental Health Flight Commander
Alcohol and Substance Abuse Prevention and Treatment Director
Chief of Psychological Services

2003-2004 ***Professional Clinical Internship:*** *Malcolm Grow Medical Center/USAF*
Rotations: Clinical Health Psychology/Behavioral Health
Life Skills Support Center/Mental Health
National Security Agency

2003-present Commissioned Officer in the United States Air Force (USAF)

2002-2003 Drexel University Student Counseling Center
Psychotherapist

2001-2002 Hahnemann University Hospital Neuropsychology Department
Neuropsychology Intern

2000-2003 Clinical Neuropsychology Associates
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